3 1761 06705175 5

Bequeathed to the Library of the
University of Toronto by
Irving Heward Cameron, Esq., M.B.
Sometime Professor of Surgery
in the Faculty of Medicine

Digitized by the Internet Archive
in 2007 with funding from
Microsoft Corporation

http://www.archive.org/details/cyclopaediaofpra15ziemuoft

Y [illegible]

MPr
Z

CYCLOPÆDIA

OF THE

PRACTICE OF MEDICINE.

ugo Wilhelm
EDITED BY DR. H. VON ZIEMSSEN,
PROFESSOR OF CLINICAL MEDICINE IN MUNICH, BAVARIA.

VOL. XV.

H. A. Wrig

DISEASES OF THE KIDNEY.

BY

PROF. CARL BARTELS, of Kiel, and PROF. WILHELM EBSTEIN, of Goettingen.

Translated by
REGINALD SOUTHEY, M.D., Oxon., of London,
and
ROBERT BERTOLET, M.D., of Philadelphia.

ALBERT H. BUCK, M.D., NEW YORK,
EDITOR OF AMERICAN EDITION.

297274
3.34

NEW YORK:
WILLIAM WOOD AND COMPANY,
27 GREAT JONES STREET.
1877

Entered according to Act of Congress, in the year 1877, by
WILLIAM WOOD & CO.,
In the Office of the Librarian of Congress, at Washington.

ALL RIGHTS RESERVED.

John F. Trow & Son,
PRINTERS AND BOOKBINDERS,
205 *to* 213 *East Twelfth Street,*
NEW YORK.

BIOGRAPHICAL SKETCHES OF THE AUTHORS.

Carl Bartels was born on the 25th of September, 1822, on the little landed estate of Meilsdorf, in Holstein. His father, a farmer and lessee of the property, provided him with instruction at home in Latin, Greek, and the ordinary studies of the schools, until he was sixteen years old. Owing to insufficiency of means, however, he was unable to give him a thorough education, and the young man found himself unwillingly obliged to become an apprentice to a neighboring farmer.

During this time, having no money to provide a horse, he was obliged to do his field work with his own hands. After a three years' apprenticeship he obtained a position as clerk upon a large estate in the neighborhood of the city of Altona. Here he made the acquaintance of some advanced students in the gymnasium of the city. Association with these earnest young men revived his fondness for a scientific career, and, as his position on the farm left him leisure enough, with the assistance of a young friend, he recommenced his studies, and was so far successful that, after four years, he was pronounced ready to enter a university. At Easter, 1845, he entered the University of Kiel, and a year later went to Heidelberg, where he continued his studies in anatomy, chemistry, and physics, attended the lectures of Henle on physiology, and was introduced by Pfeufer, Chelius, and Naegele into the practical branches of medicine. His brilliant instructor, Henle, however, made the most decisive and lasting impression on his entire scientific development. In the autumn of the year 1847 he returned to Kiel, where he devoted special attention to B. Langenbeck's clinic.

When, in the spring of 1848, the first Schleswig-Holstein war against Denmark broke out, he entered the Schleswig-Holstein army as a musketeer, and served as a combatant through the first campaign. After the armistice of Malmoe, in the autumn of the same year, he received a furlough, returned to Kiel, and spent the winter in preparing himself to pass his doctorial and state examinations. This had been begun and almost completed in March, 1849, when the war again broke out, and he was recalled to the army. As there was a great lack of medical men in the field, he was given the position of sub-surgeon. In the preceding winter he had found occasion to become acquainted with Professor Stromeyer, who had succeeded B. Langenbeck in the chair of clinical surgery at the University of Kiel, and at the same time had been nominated surgeon-general of the army. Through the kindness of the latter he was ordered to the military hospital in Christiansfeld immediately after the outbreak of hostilities. Here he had ample opportunity to acquire a knowledge of the course, results, and treatment of gunshot wounds.

After the armistice, in the autumn of 1849, he was associated with Esmarch, Stromeyer's second assistant, at the surgical Clinic in Kiel. During the winter of 1849 and 1850 he passed his state examination. In the commencement of the third campaign against the Danes, in the summer of 1850, Stromeyer ordered him to the hospital in Schleswig, whither, after the fight at Idstedt, so disastrous for the Schleswig-Holsteiners, great numbers of wounded were carried. Here Bartels fell into Danish captivity, but was released two months later. Until the dissolution of the Schleswig-Holstein army, in 1850, he was employed in Kiel at various hospitals, mostly with the care of syphilitic patients. In December, 1850, he obtained the degree of Doctor of Medicine, and his inaugural dissertation was "De conjugatæ veræ pelvis introitus mensuris et mensurationibus." Interest in this theme had been excited by that excellent obstetrician Michaelis, whose clinic in Kiel he attended, and where, after his death in 1848–9, while preparing for examination, Bartels had acted as provisional assistant. In the spring of 1851, Frerichs assumed the charge of the Medical Clinic in Kiel. Bartels was an assistant, whom he honored with the task of supervising his work then to appear on Bright's disease of the kidneys. When Frerichs left Kiel, Bartels remained as assistant at the Medical Clinic, which was at first under the provisional care of F. Weber, Professor of Pathological Anatomy, and later under Professor Goetz. In company with Professor Weber he made a trip to Vienna in the autumn of 1853, and there they remained several months, pursuing studies in pathological anatomy and in skin diseases. In the autumn of 1854, Bartels resigned his position as clinical assistant. As early as 1852, he had been installed as private instructor at the University of Kiel, and gave practical instruction in auscultation and percussion. He also devoted himself to private practice, which in a measure withdrew him from scientific work. A year later, in the summer of 1858, Professor Goetz, the director of the Medical Clinic, died. Owing to this sudden and unexpected event, the authorities of the University found themselves obliged to turn over the provisional charge of the Clinic to Bartels, because he was familiar with the affairs and interests of the Institute, at which he had so long been engaged. The plan he adopted in carrying out practical exercises in the Clinic—thorough catechism—pleased the students, and when they found that they were acquiring more information by this method of instruction, somewhat tiresome at first, than by lectures simply, they joined in a general request to the Danish Ministry for Holstein, asking that he be appointed professor and director of the Medical Clinic. This request was sustained by the medical faculty. On the 1st of June, 1859, Bartels was appointed Ordinary Professor at the University of Kiel, and director of the Medical Clinic, and he holds the position to-day. His literary work has been confined to a few communications and brochures in various periodicals.

They are as follows :

Contributions of cases illustrating—1. The Theory of Bright's Disease. Deutsche Klinik. 1852.

2. Report on an Epidemic of Measles in the Spring of 1860 in Kiel. Virchow's Archiv. Bd. 21.

3. On a case of Cystinuria. Ibid., Bd. 26.

4. Pathological Investigations. Greifswalder Medicinischer Beiträge. Bd. 3.

5. Researches on the Causes of Increased Uric Acid Deposit in Disease. Deutsches Archiv. f. klin. Med. Bd. 1.

6. On Membranous Croup. Ibid., Bd. 2.

7. On the Operative Treatment of Inflammatory Exudations in the Pleural Cavity. Ibid., Bd. 4.

8. A case of Echinococcus beneath the Dura Mater Spinalis. Ibid., Bd. 5.

9. On Systolic Vascular Murmurs in the Lungs. Ibid., Bd.

10. On Peripleuritic Abscesses. Ibid., Bd. 13.

11. Clinical Studies on the Chronic Diffuse Inflammations of the Kidney. Volkmann's Samm. klin. Vorträge. No. 25.

12. On the Treatment of the Febrile Condition by the Methodical Derivation of Heat. Mittheil. f. d. Ver Schles.-Holst. Aerzte. Heft I.

13. On our Present Knowledge of Syphilis. Ibid.

14. On the Dilatations of the Stomach and their Treatment. Ibid., Vol. III.

15. Albuminuria as a Symptom of Disease. Ibid., Vol. IV.

16. The Normal Temperature of the Human Body, etc. Schriften des naturwiss. Ver. f. Schleswig-Holstein. Band 1.

17. The Air which we Breathe. A popular lecture delivered in Kiel in the year 1862.

Wilhelm Ebstein was born on the 27th of November, 1836, at Jauer, in Silesia. From 1855 to 1859 he studied medicine in Breslau and Berlin, and in 1861 obtained a position as physician in the All Saints Hospital at Breslau. In the year 1863 he received the position of prosector. In 1869 he was installed by the Breslau Faculty as a private instructor. In 1870 and 1871 his work was interrupted by the French war, but, after his return from France, he resumed private instruction, and assumed the charge of the medical department of the Breslau Almshouse. In the autumn of 1874 he was called to Goettingen as Professor of Medicine, and is now in charge of the Medical Polyclinic.

His inaugural dissertation was "On the Microscopical Changes which Amylaceous Matters Undergo when Mixed with the Fluids of the Mouth." Berlin, 1859. His habilitation essay was "On Relapses of Typhus." Breslau, 1859. Besides these, thirty-five larger or smaller articles on matters of anatomical, pathological, clinical, and physiological interest, have been published in the following journals:

1. Reichert and Dubois-Reymond's Archiv f. Anat. and Phys. Jahrg. 1864 u. 1868.

2. Max Schultze's Archiv f. mikrosk. Anat. 1870 (Glands of the Pylorus).

3. Pflueger's Archiv d. Physiologie. Band III. VI. and VIII. (Physiology of the Gastric Mucous Membrane.)

4. Virchow's Archiv f. Path. Anat. Band 34, 40, 49, 51, 55, 62.

5. Archiv d. Heilkunde. Band VIII. IX. X.

6. Deutsches Archiv f. klin. Medicin. Band 6, 9, 10, and 11, 12.

7. Archiv f. experim. Pathologie. Band II.

8. Wiener med. Presse. 1865, 1866, 1869.

9. Berliner klin. Wochenschrift. 1873, 1874, and 1875.

10. Berichte der deutschen-chem. Gesellschaft in Berlin. 1875. He has also published an article on Carcinoma of the Stomach in "Volkmann's Sammlung klinischer Vorträge."

EDITOR'S NOTE.

Through an oversight on the part of the editor, the translation of the entire article on Syphilis of the Brain and Nervous System, in Vol. XII., was credited to Dr. Robert T. Edes. The portion relating to Syphilis of the Brain was translated by this gentleman, while that relating to Syphilis of the Spinal Cord and Peripheral Nerves should have been credited to Dr. Louis Velder.

CONTENTS.

BARTELS.

PAGE
STRUCTURAL DISEASES OF THE KIDNEY AND THE GENERAL SYMPTOMS OF RENAL AFFECTIONS 3
Local symptoms 4
Pain 4
Inspection 5
Palpation 6
Percussion 8
Functional symptoms, which are proffered by the quantity and quality of the urine 10
Quantity 12
Color and other general features 17
Specific gravity 19
Chemical composition in diseases of the kidney 23
Reaction 23
Urea 26
Albuminuria 30
Chyluria 32
Doctrine of albuminuria 34
Hæmaturia 66
The formed elements of the urine in renal disease 72
Urinary casts or cylinders 75
Disorders manifested by the blood, by the general nutrition, and especially by the nervous system, when the kidneys cease to perform their functions properly 96
Dropsy that accompanies renal diseases 96
Uræmia 109
Convulsions 109
Amaurosis 110
Mental and digestive disturbances 110
Respiratory disturbances 111
The causes 112
Symptoms due to disturbances of the general nutrition produced by renal diseases 150
Dyspepsia 151
Vomiting 152

PAGE
Diarrhœa 154
Hemorrhages 156
THE DIFFUSE DISEASES OF THE KIDNEYS 163
History 163
Classification 189
Hyperæmia 190
Active hyperæmia 190
Etiology 191
Anatomical changes 192
Symptomatology 192
Diagnosis 194
Treatment 196
Passive hyperæmia 197
Etiology 199
Heart diseases 199
Lung diseases 200
Pathological anatomy 201
Symptomatology 204
Course and terminations 213
Diagnosis 214
Prognosis and treatment 216
Ischæmia of the Kidneys and its Results, The Choleraic Affections of the Kidneys 222
Etiology 222
The morbid anatomy of the kidney in cholera 226
Pathology 229
Symptomatology 233
Diagnosis and prognosis 242
Treatment 243
Acute Parenchymatous Nephritis 244
History 244
Etiology 246
General summary of course and symptoms 262
Pathological anatomy 265
Symptomatology 273
Prognosis 288
Diagnosis 292
Treatment 295
Acute Parenchymatous Nephritis of Pregnancy 304
Etiology 304
Course of the disease 311
Pathological anatomy 314
Symptomatology 316
Prognosis 322
Diagnosis 323
Treatment 323

PAGE
Chronic Parenchymatous Nephritis ... 324
Etiology ... 325
General course of the disease ... 336
Pathological anatomy ... 343
Symptomatology ... 353
Duration ... 380
Prognosis ... 381
Diagnosis ... 393
Treatment ... 397
Renal Cirrhosis ... 408
Etiology ... 409
Progress of the disease ... 415
Pathological anatomy ... 451
Analysis of the symptoms ... 462
Retinitis albuminurica ... 476
Duration of genuine contracting cirrhosis of the kidneys ... 480
Prognosis ... 482
Diagnosis ... 484
Treatment ... 485
Amyloid Degeneration ... 496
History ... 496
Etiology ... 498
Course of the disease ... 504
Duration ... 516
Pathological anatomy ... 517
Analysis of the symptoms ... 523
Prognosis ... 529
Diagnosis ... 533
Treatment ... 536
Complications of amyloid degeneration of the kidney with chronic parenchymatous nephritis and with contracting kidney ... 538

(Translated by Reginald Southey, M.D.)

EBSTEIN.

DISEASES OF THE KIDNEYS, TOGETHER WITH THE AFFECTIONS OF THE PELVES OF THE KIDNEYS AND THE URETERS ... 543
Inflammations of the Kidney, of the Pelvis of the Kidney, and of the Perinephritic Tissues, with Termination in Suppuration ... 543
Suppurative nephritis, renal abscess ... 543
Bibliography ... 543
Introductory remarks and history ... 544
Etiology ... 545
Pathological anatomy ... 547
Symptomatology ... 552

PAGE

Complications 560
Diagnosis 560
Duration, termination, and prognosis 561
Treatment 561
Pyelitis and pyelonephritis 562
Bibliography and history 562
Etiology 562
Pathological anatomy 566
Symptomatology 570
Diagnosis 574
Duration, termination, and prognosis 575
Treatment 575
Nephrophthisis. Caseous inflammation of the kidneys, the renal pelves, and the ureters 577
Introductory remarks and history 577
Bibliography 578
Etiology 578
Pathological anatomy 579
Symptomatology 581
Diagnosis 584
Duration, course, and prognosis 585
Treatment 586
Peri- and paranephritis 586
Definition, history, and bibliography 586
Etiology 587
Pathological anatomy 590
Symptomatology 593
Complications and sequelæ 597
Diagnosis 597
Duration, termination, and prognosis 600
Treatment 601
Degenerative Processes of the Kidneys 602
Cloudy swelling and fatty degeneration 602
Bibliography 602
Etiology 602
Pathological anatomy 603
Symptomatology 604
Hemorrhagic infarction of the kindeys 604
Etiology 605
Pathological anatomy 606
Symptomatology 606
Course, prognosis, and treatment 607
Necrosis of the kidney 608
Etiology 608
Pathological anatomy 608
Amyloid degeneration of the kidney, lardaceous or waxy kidney 609

PAGE
Bibliography 609
History 610
Etiology 612
Pathological anatomy 616
Symptomatology 624
Complications and sequelæ 629
Diagnosis 630
Duration, termination, and prognosis 631
Treatment 632
Tumors of the Kidney, of the Pelvis of the Kidney, and of the Perinephritic Tissue 635
Bibliography and introductory remarks 635
Renal cysts and cystic degeneration of the kidneys 638
Bibliography 638
Cyst-formations in extra-uterine life 639
Congenital cysts 641
Hydronephrosis 643
Bibliography 643
History 644
Etiology 644
Pathological anatomy 648
Symptomatology 651
Complications and diagnosis 654
Duration, termination, and prognosis 656
Treatment 656
Cancer of the kidney 658
Bibliography and history 658
Etiology 658
Pathological anatomy 662
Symptomatology 670
Complications 677
Diagnosis 678
Duration, course, and prognosis 683
Treatment 684
Lymphatic neoplasms 685
Tubercles of the kidney 685
Bibliography 685
Pathology 686
Foreign Bodies in the Kidney, the Pelvis of the Kidney, and the Ureters 687
Nephrolithiasis 687
History and bibliography 687
Pathological anatomy 694
Deposits and concretions in the kidneys, the renal pelvis, and the ureters 694
Pathological changes produced in the kidney, the pelvis of the kidney, and the ureters, by renal concretions 705

PAGE
Symptomatology........ 708
Complications and sequelæ........ 720
Diagnosis........ 724
Duration, termination, and prognosis........ 728
Treatment........ 730
Animal Parasites of the Kidneys........ 742
The echinococci of the kidneys........ 742
Bibliography and history........ 742
Etiology........ 743
Pathological anatomy........ 745
Symptomatology........ 748
Diagnosis........ 752
Duration, course, and prognosis........ 755
Treatment........ 756
Strongylus gigas (Rudolphi)........ 757
Bibliography and general description........ 757
Pentastomum denticulatum........ 758
Distoma hæmatobium (Bilharz)........ 758
Bibliography and general description........ 758
Spiroptera hominis and Dactyleus aculeatus........ 760
Anomalies in the Position, Form, and Number of the Kidneys........ 761
The fixed dislocation (dystopia) of the kidneys........ 761
Bibliography........ 761
General description........ 762
The movable (migratory) kidney........ 763
Bibliography and history........ 763
Etiology........ 764
Pathological anatomy........ 767
Symptomatology........ 769
Diagnosis........ 772
Duration, course, and prognosis........ 774
Treatment........ 774
Anomalies in the form and number of the kidneys........ 776
Bibliography........ 776
The lobulated and the horse-shoe kidney........ 776
Absence of the kidney........ 778
Diseases of the Renal Vessels........ 780
Bibliography........ 780
Diseases of the renal artery........ 780
Pathology........ 780
Diseases of the renal vein........ 781
Etiology........ 782
General description........ 783

(Translated by Robert Bertolet, M.D.)

THE
STRUCTURAL DISEASES OF THE KIDNEY,
AND
GENERAL SYMPTOMS OF RENAL AFFECTIONS.

BARTELS.

THE

GENERAL SYMPTOMS OF RENAL DISORDERS.[1]

THE symptoms that mark the progress of disease situated in the organs set apart for the elimination of urine fall into three great natural groups.

The *first group* comprehends the local abnormal phenomena situated in and strictly confined to the region of the kidneys. These too may be separately considered under two heads: first, as mere subjective sensations, the sense of pain or discomfort which a patient experiences; and, secondly, as objective facts, alterations either in the situation or in the size, which can be felt and seen, or in the consistence of the organs themselves.

The *second group* of symptoms comprises those which bear upon the performances of the kidney as an excretory gland—the character of the urine secreted, its departure from the normal standard in quantity or quality—the functional symptoms really of greatest moment towards a correct diagnosis.

To the *third group*, finally, belong the general constitutional symptoms, the widespread mal-nutrition, and disordered functions which notify the effect upon the system at large, and especially upon the nervous system, of an imperfect depuration of the blood, and a generally lessened activity of the whole body.

---

[1] For references to authors, look to foot-notes at the bottom of the text; the shortness of time allowed him for writing this work not having permitted the author to append a complete catalogue of the literature of his subject.

I. Local Symptoms.

1. Foremost among the *subjective sensations*, we may reckon pain situated in the region of the kidney. Whether the kidney itself is furnished with nerves of common sensation, is as yet undecided; but this much we know, that considerable pathological changes may progress in the substance of these organs, and be totally unaccompanied by pain. On the other hand, we are aware that considerable suffering has been endured when the renal capsules have either been implicated by inflammation or distended in consequence of swelling of the gland or growth of new formations in it, and that irritation of the nerves supplied to the pelvis of the kidney or to the ureter is apt to be exceedingly painful.

Patients usually describe what they feel as a dull sensation of weight or tension localized about the loins, on one or sometimes upon both sides of the spine, and, if pressure be made in this locality, whether from behind in the lumbar region, or through the abdominal walls, from in front, some tenderness is generally to be detected in the same situation. A further feature of this pain is its exacerbation by any blow or shake of the body in running or moving about; and there is this peculiarity about the kidney pains, which has not failed to attract attention, namely, that they are very often referred to the groin, that they are reflected along the distribution of the crural nerves, and that in males they run down the spermatic nerves, producing a contraction of the cremaster muscle and retraction of the testicle close up to the inguinal canal. Sharp pain in the kidney is often attended by reflex vomiting, and not uncommonly leads to sudden attacks of faintness as well as other symptoms of general collapse. Pain, however, of this intense kind—renal colic, as it is called—is nearly only produced by renal calculi, and is paroxysmal, paroxsyms of pain alternating with certain free periods of longer or shorter duration.

In by far the larger number of kidney diseases proper, pain is either absent altogether or transitory, and of quite trivial importance; severe and enduring pain has been observed by me only in the traumatic injuries of these organs, after crushings, from abscess, or in the course of cancerous disease, while in each and every instance it was quite impossible to determine whether the pain arose from the nerves of the kidney, or from those supplied to its capsule, or from branches distributed to affected organs in its immediate neighborhood.

2. The *local objective symptoms admitting of demonstration* are really fewer in affections of the kidney than in those of almost any other organ in the body, since these glands are situated so deeply in the abdominal cavity upon either side of the spine as to be very difficult of access. Concealed as they are by the thickness of the abdominal walls, covered at least in part by the lowest ribs, arched over by the transverse processes of the vertebræ, and hidden by the muscles of the back and musc. quadrat. lumborum, it is little to be wondered at that the exact position, size, or state of the kidneys, whether indurated or otherwise, should be difficult to determine. Slight alterations in their shape or consistence will escape the observation of the physician altogether, and in fat subjects any local exploration would be a work of supererogation.

When one of the kidneys is converted into a tumor of any considerable size, or presents its pelvis distended into a large cyst, one is often able, by mere *inspection* of the body, to notice the protrusion of the affected side; but should a similar enlargement be present in both kidney regions, this would admit of much less definite demonstration. The site usually occupied by such a tumor is the side of the body between the lower border of the twelfth rib and the brim of the pelvis. In this situation the abdominal walls may be bulged out before it, the lowest ribs being occasionally protruded so much that the circumference of the thorax upon the affected side is notably increased. The renal tumor usually enlarges from behind forwards, extending towards the umbilicus.

While the rule certainly obtains that renal tumors are usually most prominent in the side and back, examples no doubt occur in which enlargements of the kidney are directed more towards the front, approaching the anterior wall of the abdomen. Thus

I have seen a case of cancer of the left kidney which protruded as a palpable mass between the ribs and the median line of the body above the umbilicus.

An œdematous condition of the skin and subcutaneous tissues occurring between the lowest rib and the brim of the pelvis, and reaching posteriorly towards the spine, will sometimes betray a perinephritic abscess, a process of suppuration which generally takes its origin in inflammation either in the substance or in the pelvis of the kidney.

In a very emaciated woman, who had gone through several confinements, I once made out a prominent tumor in the right iliac region, that I could recognize distinctly by its shape as a displaced or floating kidney; it further admitted of being easily put back into its normal situation.

Of far more use, however, than mere inspection in the diagnosis of diseases of the kidney, comes the *examination* of the renal region *by the hand.* By careful manipulation one is often enabled to determine, not merely displacements and alterations of size, but sometimes the actual consistence of those parts of the organ which are accessible to touch.

Still it is only in a limited number of instances that one arrives at exact knowledge in this way; in far too large a number of cases we obtain no information whatsoever; indeed, in only one out of all the examples of parenchymatous inflammation of the kidney which I have seen, where the patient, a woman, was pretty thin, was I able to make out by my fingers through the abdominal walls that both kidneys were swollen, and to arrive at even an approximate estimate of the enlargement of these glands, which must have been considerable. On the other hand, I have never been able to discover any atrophy of the organs, since the natural-sized and normally situated kidney is in nearly every instance inaccessible.

Palpation, then, as an aid in diagnosis is of special value in discovering renal tumors and cyst-like distentions of the pelvis of the kidney.

These tumors and cystic enlargements certainly, as a rule, occupy that space which lies between the twelfth rib and the brim of the pelvic bone and fill it completely, right back towards the spine; while enlarging, as they mostly do, from behind forwards, they push out the abdominal walls before them.

As before remarked, however, there are exceptions to this more usual course of things, and the exceptions are, for the most part, cases in which the entire organ is not equally enlarged in every direction; this obtains illustration from the example already cited by me, in which a cancerous tumor of the left kidney advanced forwards between the linea alba and the left arch of the ribs, from beneath the left hypochondrium, above the umbilicus, quitting the left lateral abdominal or lumbar region altogether.

Cystic tumors formed by retention of fluid in the pelvis of the kidney also do not always occupy the left lumbar region, but push themselves so far to the front that a coil of intestine may lie behind and outside them.

The general rule, however, obtains, that kidney tumors may be sought for in the back and flanks; and here it becomes important to notice that renal tumors occurring upon the left side, and attaining to any considerable size, are often crossed by the descending colon, which may be felt as a tough cord, traversing them obliquely from above downwards and from without inwards.

In such a case, the tumor, from being situated behind and outside the peritoneum covering the anterior surface of the colon, will drag this portion of peritoneum some distance away from the posterior abdominal wall, so that the large intestine, together with this bit of peritoneum, will form part of the anterior sheath or casing of the tumor.

Rosenstein correctly enough insists, however, that this relation of the great gut to left-sided kidney tumors is by no means an invariable one.

Palpation, then, not only gives us information of the size, shape, and consistence of renal tumors, but enables us to determine their relational anatomy. We may discover the hardness, and in many cases the knobby, uneven surface of cancer of the kidney, and may decide upon the existence of fluctuation appointing a single large sac with fluid contents—a hydronephrosis; but it more rarely happens that we are able to make out that cystic degeneration of the substance of the kidney, in which cysts of considerable size may be present, or to discover the segmentations with their several prominences appointing different sacculations that may compose one huge kidney tumor, or even to make out fluctuation in them.

Very characteristic, however, is the wooden hardness and sharp definition that distinguish the lumbar tumor, and indicate the presence of products of inflammation infiltrating the tissues round about a kidney, and equally significant is the doughy, œdematous state of the integuments and abdominal walls, which usually accompanies the same event. In an examination, in which we employ our hands only, we may be able to speak positively to a kidney being preternaturally movable and displaced, while both from the shape and from the mobility of some solid organ, which admits of being easily shoved in and out of its natural resting-place, we can arrive at the diagnosis of a floating kidney.

The local exploration of the kidney will be best attained if the patient be directed to lie upon his back, with the thighs slightly bent upon the trunk, and at the same time separated somewhat from each other; in this posture the abdominal muscles are best and most easily relaxed; then the physician should place himself upon that side of the bed and body of the patient which he is desirous of investigating, looking him face to face.

Thus, if the tumor be in the right abdominal region, he should be on the right side; let him then place his right hand upon the anterior wall of the abdomen, and pass his left hand to the back of the patient, pressing the kidney region in the loins with his left hand from behind forwards, inserting his fingers, so far as he is able, between the lowest rib and the pelvis, and pushing any tumor forward against the palm of the right hand.

Far less valuable, because less reliable, information is obtained from *percussion* than from palpation in kidney affections. For the purpose of making a proper examination by percussion, the patient must be laid upon his belly, with a pillow beneath this, in order that the back may be well stretched. There exists normally a space about five centimetres broad in the lumbar region, between the twelfth rib and the brim of the pelvis, which gives a dull note when sharply struck with a hammer upon a plessimeter, and if the plessimeter be moved towards the anterior abdominal wall, this dullness is quickly exchanged for tympanitic resonance, the latter marking the course of the intestine which borders against the kidney.

The dullness marks the situation of the organ surrounded by its natural fat envelope; but neither upwards nor outwards can its limits be more definitely explored, since this dullness merges into that due to the liver upon the right, and into that due to the spleen upon the left side, nor can its boundaries in a downward direction be more distinctly set, since the lower end of the kidney lies, as a rule, within the pelvis, and is completely removed from exploration by percussion by the intervention of the pelvis with its thick muscular coverings.[1]

Now, if a kidney be at all notably enlarged, the area of dullness marking it will extend further outwards than it ought to. Should any considerable tumor, like a hydronephrosis, be present, the dullness when the patient lies on his back may reach forward towards the navel, aye, and beyond it, too, right over to the opposite side of the body; thus it happens, in consequence of the ascending colon traversing this tumor upon the right, and the descending colon upon the left side, that, according as the portion of intestine is full or empty of gas, the resonance will vary, being increased or diminished in breadth correspondingly.

On the left side the colon passes, as previously described, from above downwards and from without inwards, while upon the right side, as Rosenstein has observed, the cæcal appendage and lower portion of the ascending colon may lie outside the tumor, and, dividing the kidney from the liver, separate by a band of resonance the dullness due to both these organs.

Very large renal tumors may push either liver or spleen with its diaphragm covering high up into the thoracic cavity, and thus lead to complete alteration of the normal chest tones.

---

[1] My friend and colleague, Heller, has been good enough to examine a number of bodies for me, with the view of determining the ordinary situation of the kidneys. He found that the lower end of both organs came down, almost without exception, below the brim of the pelvis.

Then, too, as Gerhardt has shown, cases present themselves in which dullness on percussion leads to the discovery of a renal tumor even before we can feel it.

Inflammatory infiltrations into the cellular tissue round the kidney increase its area of dullness just in the way a tumor might. General swelling of the kidney cannot, however, be determined by percussion with any certainty; at least, I have never arrived at thus discovering it; in fact, our endeavor to make this out would be frustrated—first, by the general anasarca of the tissues of the back, the usual accompaniment of serious glandular nephritis, and, secondly, by the ascites so common in this affection. Atrophy of these organs does not admit of demonstration by percussion any better.

When a kidney is displaced, there will be resonance over its normal site, and dullness again upon its replacement in loco naturali; similarly, the dullness proper to it will be altogether lacking, if the kidney on either side is absent.

In fat subjects, percussion conveys no information whatever, since the accumulation of fat in the retro-peritoneal connective tissue will lead to dullness far out from the vertebral column.

II. The Functional Symptoms, which are proffered by the Quantity and Quality of the Urinary Secretion.

In testing the functional capacity of the kidneys, whether for physiological or for diagnostic purposes, one must bear in mind the fact that the secreting energy of these organs depends upon constantly varying factors, and that therefore both the quantity and the composition of the secretion will be subject to corresponding variation. Neither the specific gravity, nor indeed any other feature of a urine passed on one particular occasion, is invariable. The sample passed at one time may differ from what preceded or succeeded it.

This physiological fact will help us to a better understanding and more correct interpretation of the pathological processes in kidney diseases. It happens that, in a good many renal disorders, we can only arrive at a correct diagnosis, and furnish an opinion upon the probable progress and ultimate issue of the case, after making a protracted and most careful examination of the urine, and after instituting this scrutiny into its quantity and other special characters over and over again.

The analysis that is to enable us to surmise the existence of many pathological states, to refer the symptoms to their kidney origin, and in not a few cases to predicate danger before this has arisen, must be thorough of its kind.

Here, then, is the first rule, which must be scrupulously laid down for the guidance of any one who desires to vouchsafe a correct diagnosis in any renal disease, namely, that the observer must rest satisfied that he has secured for his analysis the entire urine passed by his patient. *It must be the total urine of twenty-four hours, scrupulously collected together.*

I well know how difficult it is to arrange this, even in a hospital where the nursing is good; and I am aware how prone attendants are to deny that there has been any loss by spilling, and to keep silence when, by some carelessness of theirs, a specimen of urine has been spoilt for analytical purposes.

The object of the medical man will be most surely attained, and fewest mistakes will happen, if he can succeed in convincing the patient of the importance of aiding him in his task. Especial pains must be taken to guard against any urine being lost at stool, and some mechanical contrivance should be adopted rendering the separate collection of the urine passed on these occasions feasible.

The samples required for separate examination are to be taken from the total excretion of the twenty-four hours collected together. Within some period of about this duration, all those various causes that do affect both the rapidity of secretion and the composition of the urine passed at different periods of the day, will have come round again in a regular and ordinary way.

It is only in pursuit of some very special object that this ordinary rule in making a diagnosis can be departed from, since in no shorter period than this is it possible to obtain a urine that furnishes anything like a correct sample of the kidney's performances, and to determine how much urinary water, how much nitrogenous material, as well as how much albuminous substance, are being daily excreted.

An examination of the urine must include a notice of its *quantity*, *color*, *general appearance*, *specific gravity*, *chemical composition*, *and the characters of its sediment.*

It cannot be my task in this place to enter into any description of the manner in which these various inquiries should be carried out. The work is one which requires special teaching and practice; and we possess, in the manual of Neubauer and Vogel, upon "Qualitative and Quantitative Urinary Analysis," which has already run through its sixth edition (1872), a treatise which gives all the instruction requisite to its thorough performance.

1. *The Quantity of the Urine.*

The quantity of urine secreted by the kidneys in twenty-four hours is now well known to vary considerably even in healthy persons, and to vary not only with the individual, according to age and particular habits of life, but to be different in the same person under altered circumstances. The rapidity of the secretion depends on several factors: first, upon the blood-pressure in the Malpighian tufts, the parts which we are wont to regard as the sources of the urine water; although, if we expressed the matter more correctly, we ought to attribute differences in the rapidity of the secretion to differences in the pressure between the blood on the one side, contained in the capillary coils, and the fluid on the other contained in the tubuli uriniferi (Ludwig). Let us for the moment, however, regard this pressure in the renal tubules as a constant quantity, then the blood-pressure in the capillaries, from which the urine water is separated, will fluctuate with the tension maintained in the whole arterial system; and since the tension in the vessels at large must be affected by diminution or increase of their contents, so, too, the urinary excretion will vary accordingly. Common experience teaches us that when we drink more fluid than is our ordinary habit, and, by exposure of our bodies to cold and wet atmospheres, diminish the watery elimination taking place through the skin and lungs, our urinary secretion is noticeably increased in quantity; and, on the other hand, we know that we are able to reduce the quantity of urine water secreted very considerably, by either diminishing the ingestion of fluid or directing its waste, whether by quickened respiration, or sensible or insensible perspiration, through other channels—as, for example, by purging, when huge quantities of watery fluid can be drained away from the system. The profound influence of profuse sweating in determining the quantity of urine excreted in renal disease is perceived in the course of any of the febrile disturbances which are apt to complicate it.

Less well understood are those influences exerted from without through the nervous system upon the urine secretion, acting as they do under both normal and abnormal conditions of life in promoting or arresting the excretory energies of the kidneys. Still, the power of nervous emotions in this direction is placed beyond all doubt, both by physiological experiment and by clinical experience.

In any case, in considering what influence the nerves may have upon the quantity of the urine, we have to reckon not merely the direct stimulus which is conveyed to the walls of the blood-vessels, and which thus regulates the blood-pressure in the capillaries of the Malpighian bodies, but also the fact, now established by physiologists for the kidneys as certainly as for the salivary glands, that these organs are provided with special nerves ministering to the function of secretion.

These nerves betray their action and influence to us physicians often enough, and I need only ask you to remember how fear and high emotional tension quicken the urine flow, and conversely how hysteria will sometimes lead to a nearly complete suppression of the urine.

Since Mosler[1] showed, in so many instances, the effect which damage of particular portions of the nerve-centres had in producing anomalies of secretion, I have been in the habit of regarding every case of so-called diabetes insipidus as the consequence of disease in, and perverse activity of, the special secretory nerves supplied to the kidneys.

---

[1] *Prof. Fr. Mosler*, Neuropathische Enstehung der einfachen Harnruhr (Hydrurie durch Meningitis cerebro-spinalis epidemica, durch Trauma, durch Syphilis). Virchow's Archiv. Bd. 58, S. 44.

Lastly, the quality of the blood, reckoning herein its several constituents, has no mean effect upon the quantity of urine daily excreted. Thus the more watery the blood is, the blood pressure remaining the same, the larger will be the quantity of fluid that the kidneys must excrete. The increased urine flow that follows upon excessive ingestion of water, and the diminished discharge succeeding such abstinence from drinking that the sense of thirst is sorely felt, are surely to be attributed in no small degree to the fluctuations of the water contained in the blood serum and to the alterations in arterial pressure thereby involved.

The actual quantity of urine, however, is determined by the special urinary constituents contained in the blood serum, and by those crystalloid substances which are capable of separation by the kidneys. According to Ustimowitsch,[1] in an animal kept alive by artificial respiration after division of the spinal cord between atlas and occiput, when the kidneys have ceased acting in consequence of the operation, urinary water can be made to flow in full stream again if urea is injected into the blood; and exactly the same result follows, according to Heidenhain,[2] if urate of soda is employed. I do not doubt myself but that the well-known diuretic action of common salt, and in part, too, the excessive urination in diabetes may be similarly explained.

Albeit the daily quantity of urine then does vary in sound, healthy persons subjected to different circumstances, its actual fluctuation rests within certain well ascertained limits under ordinary regular habits of life, although in this, as in everything else, there are idiosyncrasies. Careful observations have enabled us to fix a certain mean average diurnal urine excretion for any individual, and this Vogel calls his "individual urinary capacity."

In practice we are compelled to arrive at the normal urinary capacity of a patient at the time when we see him, and when he is ill, and when we have only a urine which is itself a departure from the normal to guide us, and no observation upon his urine in health to refer to and correct ourselves by. For him we must assume a normal mean average, and to this end we take what has been established for sound persons by repeated observations (Vogel).

---

[1] Leipziger Berichte vom 12. December, 1870. (I am obliged to quote from Heidenhain, for I have not the original by me.)

[2] Versuche über den Vorgang der Harnabsonderung. Pflüger's Archiv für die gesammte Physiologie. Bd. IX. S. 26.

Different authors are not exactly agreed as to what this normal mean average urine quantity for the twenty-four hours should be.

According to Vogel, the mean average quantity for a thoroughly sound adult may be reckoned as follows :

In twenty-four hours.

1,400 to 1,600 cubic centimetres for *persons who drink freely.*
1,200 to 1,400 " " " *those who drink moderately.*

In one hour.

50 to 70 cubic centimetres for *free drinkers.*
40 to 60 " " " *moderate drinkers.*

He calculates that a healthy adult ought, *on an average, to excrete per hour one cubic centimetre of urine for each kilogramme of weight of his body*, and, according to the same observer, the daily quantity of urine passed by a person of ordinary health, but living an irregular life, may fluctuate between one thousand and three thousand c. ctm., and the hourly quantity between twenty and two hundred c. ctm.

When the kidneys are affected by disease, a multitude of different factors come into action, which produce greater or less excretory energy in these glands and determine considerable departures from the normal in the secretion furnished by them, in respect both to quantity and to quality.

For example, the number of tubes or excretory channels may be diminished. Experience teaches us that nature here, as in every other part of our bodies, has endowed us with very lavish liberality, and we are aware that one entire kidney may be either congenitally defective or destroyed by disease, and the individual's health be little or no ways affected in consequence, the single organ that remains amply sufficing to fulfil the functions of two, and performing its functions thoroughly satisfactorily. For self evident reasons, no one had been able, up to quite recently, to test the excretory capacity of any such solitary kidney, for no one kidney was totally absent. But Simon has just furnished us with a case in point, in which he successfully extirpated a sound kidney on account of fistula of its ureter; this case, which was admirably observed and treated with both courage and skill by him, conclusively proves that one kidney is able to eliminate as much urine per diem as constitutes the ordinary mean average quantity of a healthy person.

The woman thus operated on passed one thousand three hundred c. ctm. of urine on the twelfth day after the operation; and although, later on, for the further six weeks she remained under observation, the quantity passed by her ranged rather below the ordinary diurnal average, it would have been impossible to regard her as otherwise than in the enjoyment of perfectly sound health.[1]

Clinical experience has taught us, too, that a loss of the glandular secreting vessels (the tubes) not only does not necessarily involve a diminution of secretion commensurate with such loss, but that contrariwise, in a great many cases where the kidneys are contracted, and considerable destruction of the Malpighian tufts has taken place, a quantity of urine which is far beyond what is normal has been secreted. I have treated a patient suffering with such an affection, who passed in twelve hours six thousand c. ctms. of urine; still I must notice here that the process of contraction in the kidneys may advance to such an extent, that what remains of the organ really is unable to separate the ordinary mean average of urine. A diminution of the daily excretion below the normal average, extending even to entire suppression, can follow as the consequence of simple alterations in blood pressure, as happens, for example, in the algid stage of cholera, before any, even the very slightest alterations have taken place in the secreting structures. It is more common, however, for such a diminution of secretion as at all approaches to suppression to depend on pathological alteration of the glandular substance, or to follow upon some block in the channels by which the secretion ought to drain away. Indeed, there is no more certain or frequent source of complete suppression of urine than the last-named event.

---

[1] The translator ventures to remark that the capacity of one single kidney to carry on the work of the economy is not to be disputed, but the entire sufficiency of such a single organ to maintain the health of an individual can be hardly held proven by the above instance. Health is always an equivocal term, and perfect health consists in a certain amplitude of structural as well as functional endowment. His own experience has taught him that single organs, performing double duties, do them, for the most part, at a cost in extra wear and tear, which becomes more and more appreciated as life is prolonged. That the mal-functioning of such an organ does not admit of being recognized at first is a little surprising, but a little later evidence would be more valuable. He would like to know how this patient's kidney bore the extra tax put upon it a year or two subsequently to the operation.

As an example of a kidney affection, which is capable of producing diminution, if not complete arrest, of secretion, let me mention diffuse inflammation of the glandular parenchyma.

Coincidently with the swelling of the kidney substance, and blocking of the renal tubes with cylindrical casts, the outflow of the urine from the excretory channels will be impeded, and the pressure of this must then extend backwards upon Mr. Bowman's Capsules (the Glomeruli); if the tension here be at all considerable, it will so far counterbalance the blood pressure in the capillary tufts as to prevent any further secretion taking place.

2. *The Color and other General Features of the Urine.*

The color and general appearance of the urine may suffer notable alteration in kidney disease; and to determine this, it is only necessary to collect it in clear, colorless glass vessels, and study these.

The rule, under ordinary circumstances, is for the color of the urine to betoken the amount of urine pigment excreted, and the quantity of this depends upon the capillary interchanges all the body over, and the destruction of red blood coloring matter involved therein.[1] The amount or depth of color corresponds to the character of the secretion. Thus the deeper colored any urine is, the smaller amount of urinary water will there be diluting it, and conversely, the paler it is at any given time, the more abundant it ought to be, because the more urine water will be mixed with it.

---

[1] The recent observations of Jaffé, Stokvis, Vierordt, and others, show that it is probable that a part, if not the greater portion, of the urine pigment, emanates from the bilirubin of the gall, or rather from the hydrobilirubin formed out of this, in the intestines; and it is quite certain that the quantity of bilirubin formed may be accepted as the measure of the amount of destruction that has taken place in the coloring materials of the blood; it is quite unnecessary, therefore, for me to examine these observations more minutely in the present pages.

The proposition holds good, too, for kidney disease, generally speaking. Only one must remember that in most renal affections the blood becomes very poor indeed, oligocythæmia prevailing for the most part, and that both poverty of red blood, as well as excess of water in the blood, will act together in determining one and the same result. Now it is owing to this that in renal disease, in spite of scanty secretion, we find the color of the urine often very pale.

In any case of hæmaturia, be the source what it may, either from the secreting or from the conducting channels, the color and aspect of the urine become all-important for diagnostic purposes. Thus if the blood be furnished in the shape of large clots that one can see at once, it may safely be vouchsafed that these could not have issued from the secreting structures. They must have joined the urine upon its passage outwards, and will have been furnished, either by the pelvis of the kidneys, or by the ureters, or by the bladder, or, lastly, they may have become incorporated with it in the urethra itself. The blood shed into the urine at the very source of this, the kidney, betrays itself by a very characteristic color that ranges between smokiness like beef-tea, and blackness, according to its quantity, and in every instance the blood-cells will be more or less burst, and robbed of their true color, if the bleeding have taken place in the kidney. The diffusion of the coloring matter of the blood through the urine, when the grosser sediment has settled down and the supernatant fluid become quite clear, may be recognized by a very unequivocal feature: the fluid is dichroistic, looks either red or green, according as it is regarded by transmitted or reflected light. It is true that, if the urine contain very little blood, this cannot be noticed. A red or brown-black granular-looking sediment is quite sure to settle down at the bottom of the vessel, if the blood content of the urine is at all considerable.

In many cases of kidney disease the urine is, as a rule, clear, having neither opacity nor noteworthy sediment, and this circumstance obtains especially in the examples of abundant or hyperexcessive urine secretion, when the actual large amount of urine water separated holds in solution all those substances which lend to normal urine its cloudiness and sediment; further, these are the very instances in which too little solid constituents and organic detritus are furnished by the kidneys, to proffer either opacity or noticeable precipitate. On the other hand, we encounter renal affections, in which, although plenty of urine is secreted, this is constantly cloudy, or exhibits thick layers of sediment after it has stood, being either mixed with blood or pus or phosphatic deposits, or containing abundant tube casts.

A urine is apt to present a cloudy aspect with great regularity, when the actual quantity of urine secreted falls below what is normal. Its opacity is due either to the presence of the ordinary solid constituents of the urine, like the urates no longer held in solution, because the urine has cooled down, or else to that of the organic formed elements (morphotic), as, for example, the red or white blood-cells, renal or other epithelium, detritus, or tube casts, as the cylinders formed in the renal tubules are usually called.

It has often happened to me to notice that the urates contained in a highly concentrated and also highly albuminous urine separate themselves somewhat peculiarly in the form of granular amorphous particles, diffused throughout the fluid; they do not sink as a sediment, leaving a moderately clear supernatant stratum above them as in non-albuminous urine, but maintain a cloudiness, which is pretty equally opaque from top to bottom. Highly albuminous urine froths up a great deal when it is pissed into any vessel, and this froth stands a long while without subsiding, and thus attracts the attention of common inobservant people.

3. *The Specific Gravity of the Urine.*

The determination of the specific gravity of the urine is of the greatest importance in the diagnosis of kidney diseases. The simplest and quickest plan of taking it is to employ the graduated urinometer, which has a quicksilver bulb below, an air-containing cavity above this, and at the top a scale fixed inside a glass-tube. This instrument, simple as it is, affords results which are fully accurate enough for the object we have in view, and the comparisons which I have instituted in examining varieties of urine, between its performances and the method of estimating by specific gravity beads, shows far too little difference to prove of any practical importance.

In reckoning the value that should attach to the specific gravity which we have found a urine to possess, one must remember that this secretion varies not in the quantity passed only, but in its composition at every occasion when it is passed, and this even under quite normal conditions, and that its specific gravity, which is the relation of the solid content to the watery solvent, will alter accordingly. This variation in the specific gravity depends partly upon the blood pressure at what time the secretion is taking place, and partly upon the amount of water contained in the blood serum, upon the quantity of nitrogenous material accumulated in the blood, and upon the functional activity displayed by the renal cells, all which will certainly vary at different times.

From these reasons it will be apparent that the urine secreted and passed at different times within the same twenty-four hours by a perfectly sound man will differ in density, and of course in specific gravity. My colleague, Edlefsen,[1] has by his ingenious experiments proved that the strata of urine, lying as they do in the full bladder of a healthy person, one superposed upon the other, differ from each other in their specific gravity in the most astonishing degree, according as they are secreted at different intervals of time, varying from 1018.5 to 1001.5.

But, as in healthy persons living regular and normal lives, the causes that affect the degree of concentration of the urine at one time or other of the day, will, subject to the same conditions, come round again to act with regularity, the same differences repeating themselves in the daily task finally completed in twenty-four succeeding hours, we become able to assume for every individual a normal urine density, just as we do a normal urine quantity, if only one reckons the quantity and density of the entire secretion of the twenty-four hours collected together, a rule which for diagnostic and comparison purposes admits of no exception.

---

[1] Zur Physiologie der Harnansammlung in der Blase. Pflüger's Archiv. Bd. 7, S. 499.

It is true enough that this normal density of urine always remains to a certain extent an individual peculiarity. The views of authors upon the normal specific gravity of the urine vary on this account very considerably. Gorup-Besanez says the specific gravity of the urine ranges between 1005 and 1030; Rosenstein allows a mean specific gravity of from 1015 to 1020 to normal urine. My own observations have taught me that under peculiar circumstances the specific gravity of a healthy person's urine may depart considerably outside the figures allotted by Gorup-Besanez; thus after excessive ingestion of fluid it may fall as low as 1002, and be raised as high as 1040, after such prolonged abstention from water as is presented in examples of persons undergoing the thirst cure.

The determination, then, of the specific gravity of the urine gives us approximately correct information as to its solid contents. Neubauer, speaking of this subject in his work (Anleitung zur qualitativen und quantitativen Analyse des Harns, S. 150), says: "Having ascertained the specific gravity, if you multiply the last three figures of this, when carried out to four decimals, by the number 0.233, the product will give pretty nearly the quantity of solids contained in each 1,000 c. ctm. of the urine." He mentions further the effect which temperature has upon the specific gravity, and refers to the experiments of Siemon, from which it appears that a urine which had a specific gravity of 1021 when the temperature was +12° Cent. (54° F.), marked only 1020 at the temperature of +15° Cent. (59° F.), and scaled as low as 1019 at +18° Cent. (65° F.), showing that three degrees difference of temperature (Centigrade) correspond to about one degree of the urinometer scale. One ought to know, therefore, for what normal temperature any instrument we employ has been graduated.

The causes which affect the concentration of the urinary secretion in health, affect it also in the diseases of the kidneys. Still, it must be admitted here that there are pathological states of the kidney which make the separation of a very heavy or sometimes of a very light, quite watery urine nearly impossible. Thus, very contracted kidneys can no longer furnish any very concentrated secretion; and, in spite of the extreme general hydræmia which prevails in chronic parenchymatous nephritis, when the kidney structures are swollen and the complaint is at its height, a urine of very high specific gravity will still be furnished.

Indeed, as a general rule, one must allow that in diseases of the kidney the actual solid content of the urine is the result far more of the conditions of pressure under which it is secreted than upon the character of the blood serum out of which it is separated; or, to put the fact differently and more correctly, that the specific gravity stands in an inverse ratio to the rapidity of the secretion (is lowest when this takes place most quickly, highest when this is conducted most slowly).

Further, one must remember that in the far advanced kidney diseases, although the secretion may be rapid, the daily quantity of urine passed need not be considerable. A very small remaining portion of kidney substance, even though it function as rapidly as it can, will finally furnish no large quantity of secretion; while the small quantity it is capable of producing—very watery, because very rapidly secreted—remains always of low specific gravity.

While, then, the specific gravity enables us to arrive at a pretty close estimate of the percentage of solids and normal constituents contained in a urine furnished by a healthy kidney, we must be prepared to observe that the albumen contained in the urine from diseased kidneys comes as a disturbing element in our calculations, affecting, as it does, the specific gravity independently of the urea and urates held in solution; and the proof of this will be shown by the urinary analyses I shall subsequently have occasion to communicate.

4. *The Chemical Composition of the Urine in Diseases of the Kidney.*

At the bedside there is no more important subject of inquiry than the chemical composition of the urine, since from this we obtain the most certain data upon which to ground a diagnosis or furnish a prognosis.

Towards the discovery and decision that some special kidney affection exists, the reaction of the urine with litmus paper becomes all-important. As a general rule, even diseased kidneys furnish urine that has an acid reaction; but, as to the degree of acidity exhibited in renal disease, we possess as yet, so far as I am aware, no comprehensive observations.

That this should fluctuate in disease just as it does in health, with changes of diet and those causes which expedite or retard interstitial interchanges, is likely enough, as, too, that it should be especially influenced by the quantity of urine secreted, being least acid when this is most abundant. Excessive ingestion of the carbonated alkalies—as, for instance, when quantities of soda or seltzer water are drunk, or alkalies are taken, combined with vegetable acids (diuretic salines)—will lead to the excretion of an alkaline urine in disease just as in health. Still, in many cases of parenchymatous inflammation of the kidney, after the urine has been persistently alkaline for months and even years, I have observed in a patient a change of reaction take place from one day to another without there being any reason to explain this symptom.

It is only quite recently that I became aware, while examining such a case, that urine just quickly passed might exhibit an acid reaction, and then after a few hours become distinctly alkaline; and I think it likely that the urea in highly albuminous urine, in this and other cases, rapidly undergoes ammoniacal decomposition, and that it is for this reason that the entire urine collected over twenty-four hours shows so constantly this alkalinity.

The chemical reaction of the urine is of the greatest moment in the diagnosis and treatment of affections of the bladder and pelvis of the kidney. When in these affections, without any medicine having been taken to explain it, an alkaline urine is passed, this is generally found to have followed some ammoniacal decomposition of the urea, and to have been caused by some mixture in the urine of products of inflammation, furnished by the mucous membrane, and likely in its turn to lead to deposits of earthy salts within the urinary passages.

The *chemical analysis of the urine*, such as is directed for purposes of diagnosis or prognosis in renal affections, is not sufficient if it determine the mere presence of any particular substance, be this normal or otherwise; it must be pursued to ascertain exactly the quantity present, and in the inquiry the rule previously adverted to must be scrupulously observed, except there be some reason to the contrary, namely, that *the analysis must interrogate the total urine of twenty-four hours.* If this analysis is to furnish valuable data towards the decision of the nature of the disease and its possible issue, it will be directed to discover, not the functioning capacity of the kidneys alone, nor how far these fulfil their physiological task as purifying agents, but also how much albuminous material, which might otherwise be of use for building up and maintaining the body, they are wasting.

In this way only is it possible to estimate the degree in which the kidneys suffice to purify the economy from its nitrogenous waste, to ascertain the special urine elements, and to determine the extent to which the general nutrition is impaired by the shortcomings of the kidney.

Arrived at the knowledge of what quantity of urea is excreted, we are in a position to recognize betimes the risks attendant on its retention in the system, and knowing the quantity of albumen contained in the urine often enables us to make an exact diagnosis of the nature of the special malady.

We have occasion only too often, in the records of cases of quite recent date, to read chemical analyses of urine which reckon its percentage composition from some particular portion quite casually taken. Information of this kind is of singularly little value, since the composition of urine passed at different times, in the course of one and the same day, will vary as widely as its quantity and specific gravity. The analysis of one individual portion of urine at most can only authorize us to state that a particular affection of the kidney is not present, and only exceptionally, and when due attention has been paid to the other symptoms of disease present, could justify the certain diagnosis of a kidney malady.

In some cases it will not even provide an answer to the question whether the kidneys are or are not the organs specially at fault. Whoever wishes to understand the progress or realize the present state of a case of kidney disease must take the trouble to inform himself by exploring daily how the diseased organs fulfil their task. Now, my own experience teaches me that a practitioner cannot devote the time requisite to the task of making a daily quantitative analysis of the urine of a patient suffering from renal disease. This, indeed, is an affair which can be conducted alone in hospitals provided with an ample staff. Experience informs us that urine from the same case of renal disease, so long as it is excreted in nearly the same daily quantity and of the same specific gravity, has its component elements mixed pretty nearly in the same relative proportions.

Most of the kidney diseases in which one can get a strict analysis of the urine are such as are chronic in their course, and of them it may be vouchsafed that there is a well-marked fixity about their symptoms, the diseased organs fulfilling their functions very much in the same way, for the most part, over an extended period of time.

What, however, we may and must ask of the doctor who has care of the patient, is:

1. That he should be in a position to furnish an estimate of the quantity of the urinary constituents of greatest importance.

2. That he shall have satisfied himself of the quantity of urine excreted in each twenty-four hours, by carefully collecting and measuring it.

3. That he take the specific gravity of the urine repeatedly.

4. That he institute a quantitative chemical analysis of the urine, so frequently as he observes any notable alteration in the quantity or specific gravity of this secretion.

Unless such care as this be taken, it is impossible to follow the malady in its progress, or give even a guess at its issue.

In the chemical analysis of the urine the substances to which we direct our special attention in kidney disease are the urea and the albumen ; the other constituents are of far less importance for diagnosis or prognosis ; indeed, those only merit consideration which tend to form concretions, and thus excite disease in the urinary passages, and may come to react secondarily upon the kidneys.

Amongst these latter substances we may mention, as normal products, uric and oxalic acids, phosphate and carbonate of lime ; and as abnormal constituents, cystine and the ammonio-magnesium-phosphate—this last coming principally as a consequence of the ammoniacal decomposition of urea within the urinary passages.

In examining urine with the object of ascertaining what substances it contains that are calculated to form concretions, attention should be directed much more to the form in which these appear than to the quantity in which they are present, since the mere quantity of them forthcoming is of small diagnostic importance. The substances, therefore, which might form calculi become the objects far more of microscopical than of chemical research.

a. The Urea.

The quantity of urea in the urine is ascertained most easily by Liebig's Titrir method. It is true, this test is not free from error, since the decomposition of the urine by nitrate of mercury precipitates other nitrogenous substances besides urea alone, and because a urine containing from one to one and a half per cent. of sodium chloride delays the reaction a little when the reagent is added. Both sources of error come to be of small importance, however, in such analyses of the urine as are made in renal disease, since in determining the quantity of urea in these cases we need not heed so much the urea as such, as the nitrogenous substances which are present, and which may be regarded as the slag or dross of the interstitial waste of the body—the materials whose retention in the system renders renal disease so dangerous; while the error emanating through excess of chlorides present is in most renal affections reduced to a minimum, because the percentage content thereof, in most renal affections, is so small as to offer no obstacle to the application of the test. Still, as a source of error, this must not pass unnoticed. As to the mode of procedure, and the care to be exercised in the employment of this test, I may refer the reader to Neubauer (S. 159, ff.).

The urine contains a varying percentage content of urea even in healthy persons. My colleague, Edlefsen, has shown that it may differ as much as two per cent. in the different strata of fluid contained in a full bladder; it thus becomes impossible to determine what quantity a healthy kidney ought to furnish, or what we ought to regard as its normal ratio. I have observed quantities as widely apart as 0.8 and 6.0 per cent., and do not doubt that 0.8 per cent. is far from expressing the lowest possible minimum consistent with health. On the other hand, when fluids have been abstained from, up to a point when really unendurable thirst has been sustained, I have never found that more than 6.1 per cent. of urea was contained in the urine. The twenty-four hours' urine of a strong, well-nourished adult, himself no great consumer of fluid, contains an average quantity of two or three per cent. of urea.

The greater the rapidity with which the urine is secreted, the larger will be the quantity of this passed, and the smaller will be its percentage content of urea. This is the rule, which obtains of diseased as well as of sound kidneys. Wherefore in renal affections, whenever the urine is abundant or super-abundant, its percentage content of urea is very small, sometimes scarcely 0.5 per cent. In other kidney diseases, when the quantity of urine is notably diminished, it is not so uncommon to find this richer in its percentage content of urea than the mean average of urines passed by healthy persons.

This percentage content of urea, then, is only valuable when ascertained of a whole day's urine; but in that case it appoints the depuratory capacity of the kidney, and becomes of great importance.

Thus we perceive how it may happen that a kidney, which is diseased, but still is able to excrete a large total quantity of urine in twenty-four hours—a urine poor enough in its percentage content of urea—manages to conceal its inaptness for its functions, and we can understand how often a urine, small in its sum total, but loaded with a large percentage content of urea, will fail to remove the nitrogenous waste that at all corresponds with the ordinary excretion of an individual living under customary conditions.

The question poses itself afresh, how much urea should and ought the kidneys to excrete in each twenty-four hours, in order that they may fulfil their function as depuratory organs completely; and again I must answer that we possess no measure of what the normal quantity should be.

The limits accorded to the interstitial changes fulfilled, once more in healthy persons, are wide enough, varying, first, with the total sum of nitrogenous materials contained in the body (individual weight); secondly, with the quantity of nitrogenous material taken as food; and, lastly, with the activity with which the processes of re composition are carried out. Very especially is the production of urea influenced by the quantity and nature of food ingested, although, even when total abstinence is maintained, healthy kidneys continue to excrete urea so long as the person lives. As, however, under moderately regular habits of life we are able to lay down, for healthy persons, an ordinarily normal mean average of urine, both as regards quantity and solid constituents, so, the conditions of nutrition remaining alike, we are able to strike an average daily quantity of urea for any individual; this mean average quantity fluctuates, however, generally within rather broad limits.

Vogel[1] states that a healthy adult, living generously, excretes the following mean average of urea in his urine:

In 24 hours, from 25 to 40 grammes; in 1 hour, from 1.0 to 1.66 grammes. Or, reckoned by each kilogramme of body weight: in 24 hours, 0.37 to 0.60 grammes; in 1 hour, 0.015 to 0.035 grammes.

---

[1] l. c., S. 337.

He quotes the observations of O. V. Franqué, according to whom a healthy man excretes the following quantities of urea daily:

Living entirely	on animal food	51–92	grammes.
“	on a mixed diet	36–38	“
“	on a vegetable diet	24–28	“
“	on a non-nitrogenous diet	16	“

For example, an individual who is fasting and taking no alcohol, may for a long time pass no more than from 8 to 10 grammes of urea per diem. A woman of twenty-eight years of age, whom I was attending in her first pregnancy for ceaseless vomiting for twenty-five days, during which time I had her urine analyzed, passed on an average only 8.84 grammes of urea daily.

Neither the absolute total quantity of urea passed daily, nor its percentage amount, gives us much help in the *diagnosis* of renal disease. At most we are enabled to say that a urine which contains a large percentage proportion of urea could not have emanated from kidneys that were excessively atrophied, since organs thus reduced might furnish a large quantity of urine-water, but never much urea.

But towards making an accurate *prognosis*, there is no information more valuable than that which is obtained de die in diem, if only due attention be paid to the general conditions of nutrition in the patient. Only one must not indulge one's self in the idea that one single determination of the quantity of urea excreted in a day will suffice for the purpose; it can be only a prolonged series of analyses that will furnish information enough upon the quantity of this substance excreted to enable one to anticipate a dangerous accumulation in the system at no very distant date.

Further, in these inquiries it is always necessary to remember that the quantity of urea furnished is not derived alone from the nitrogenous matters taken as food into the system, but is the product of capillary interchanges that take place throughout the organism, and will be increased, therefore, by any febrile disturbance that may quicken these—in point of fact, the quantity of urea excreted being considerably raised in fever when no food at all is taken.

b. Albuminuria as a Symptom of Disease.

The most important symptom towards making a diagnosis in far the larger number of cases of renal disease is the secretion of albuminous urine. Heating the urine up to boiling-point, which coagulates sero-albumen, suffices for all ordinary purposes to distinguish the albumen, if only the urine itself be acid and has been rendered clear by previous filtration. Neutral or alkaline urine must be rendered acid before the operation by addition of nitric acid. (For special advice in the examination of urine for albumen, see Neubauer, S. 65, ff.) The surest plan of determining the presence of albumen qualitatively is that advised by Panum, and cited by Neubauer, l. c., under No. 7. Heat together equal parts of urine and a concentrated solution of Glauber's-salt, after adding a liberal amount of acetic acid to the mixture. The result is a complete coagulation of all the albumen present. All the quantitative estimates of the albumen in the urine referred to in this work were accomplished by weighing the coagulated albumen when this had been dried at a temperature of 100° C., (212° F.), after having been precipitated from urine first rendered acid and then boiled, and finally washed before it was dried.

A great many of the analyses were made for me by my former assistant and present colleague, Prof. Edlefsen, and my thanks are due to him as well for them as for the other help he has given me ; I am also grateful to my assistants who have since busied themselves in making such a large number of analyses of urine for me.

In speaking of albuminuria and the value we must attach to the presence of albumen as a symptom of disease, it behooves one to remember that this substance does not only find its way into the urine as a result of pathological changes in the kidney, but may come from the pelvis of this organ, from the ureters, from the bladder, or even from the urethra itself. Albumen is furnished by these supplementary sources, either because the mucous linings of the urinary passages have become inflamed, when pus is sure to accompany it, or because some collection of matter has burst through into the urinary channels; as, for example, an abscess either in the kidney itself or in the prostate gland, etc. In all these cases the albumen met with in the urine represents only that which is furnished by the serum of the pus; and since the kidneys usually can fulfil their functions normally under the condition of things last mentioned, namely, when the pus has found its way into the urine, and been thinned of course by mixing with it, we find as a rule that the percentage quantity of albumen present is trivial, and that a sediment of pus corpuscles accompanies it, a sediment which betrays the source of the albumen plainly enough, and explains its small amount.

I have only observed very highly albuminous urine, attended by inconsiderable sediment of pus, when the urinary passages have been inflamed in persons who have either had huge blisters of cantharides applied to them, or who have kept some ointment containing Spanish fly as a dressing on their skins, with the object of keeping up an issue. In these instances the urine, besides being clear, contains so large a quantity of fibrine that partly even inside the bladder and quickly after it was passed into the chamber-pot, it formed a firm gelatinous substance, and in one such case the clots formed in the bladder made the voidance of the urine impossible without the help of a catheter.

Purulent inflammations of the urinary channels are occasionally associated with diffuse disease of the kidney structures; indeed, in some cases of this kind both affections are alike concerned in the production of albuminous urine. In such cases the microscope occasionally gives us the information we require; thus, if one finds so-called casts in the sediment, besides pus corpuscles, one may decide with certainty that the albumen contained in the urine did not emanate alone from the purulent inflammation of the passages, but was furnished, at least in some part, by some disorder of the kidney. It is not, however, always possible to find renal casts in a sediment containing pus, when the kidneys really are concerned in the production of the albumen mixed in the urine. There remains still to mention a peculiar kind of albuminuria, in which the kidneys do not seem to be affected, and as to whose exact nature we are still uninformed—the so-called chyluria (chylous urine). This remarkable disease, which we are told is common enough in some tropical regions, especially in the Brazils and in the Southern States of North America, comes very rarely under our observation in Europe. One thing, however, especially noteworthy about it, is that a large proportion of the cases which have been studied in Europe has befallen persons who were either born on the other side of the Atlantic, or had lived there the greater portion of their lives. The urine in this disease presents, on exceptional occasions, at rare and long intervals of time, an aspect that might easily be mistaken for milk, this appearance depending upon a quantity of fatty matters, stirred up into a fine emulsion, and mixed with the secretion from the kidneys ; in fact, the fat is sometimes so abundant as to form a thick cream upon the surface of the fluid.

This urinary fat, however, looked at under the microscope, does not present the form of fat cells or fat drops, such as we see in ordinary milk, but appears as a finely granular opacity that pervades the fluid, and is capable of being entirely separated from the urine by treating this with ether. In a case which came under my care, 100 c. ctm. of urine, treated in this way, furnished 1.14 grammes of solid fat after the ether had been allowed to evaporate off entirely; in the fluid from which the fat had thus been removed, 0.98 grammes of albumen were found. Now the fat furnished by urine of this kind conducts itself exactly like the fat of chyle, and hence the name *chyluria*, which has been employed to distinguish the disease. This chylous urine invariably contains a considerable quantity of albumen, and often so large an amount of spontaneously coagulated albuminous clots in the fluid as to make the whole mass set into a soft milk-white jelly, which, from the presence of a few red blood corpuscles entangled in it, may assume a rose pink color. This coagulation of the fibrine may even take place within the bladder, and offer some obstacle to micturition, quite the same result as is known to happen after cantharides poisoning.

As invariably present as the fat and albumen in this chylous urine, we find red and white blood-cells, the characteristic formed elements of chyle ; my own experience, however, tallies entirely with that of other observers, in that I have always failed to discover renal casts, which it is very rare not to find in cases of true renal albuminuria.

Both the aspect and the character of this disease vary from time to time; for months the water may be clear and transparent, and neither albumen nor fat nor any elements of the chyle be traceable in it ; at these times the patients who, so long as the chyluria lasted, were pale, thin, and good for nothing, improve in the most astonishing manner.

Thus the fluctuations in the disease prolong its course over a long period of years before the final fatal termination is reached by exhaustion. The pathologists have up to the present time proffered no explanation of this anomaly why the chyle should thus enter at one time temporarily and at another thus more persistently into the secretion of the kidneys. We have been forced to content ourselves with the following hypothesis, that in chyluria some abnormal communication must exist between the lymph and chyle vessels on the one side, and the renal organs on the other.[1] It is easy to suppose that either in the kidney itself or in the renal passages there may exist some dilatation of the lymphatics (lymphektasis), and that by rupture of the wall of one of these its contents may pass directly into the urinary passages; proof positive, however, of the correctness of this surmise has, so far as I know, never yet been furnished in a single case of chyluria. In the meantime there are observations enough of an escape of chyle as the consequence of dilatation of the lymphatics in other organs (penis and scrotum, for example). Anatomical proof has been given of the occurrence of lymphangioma in the kidneys,[2] so that the conjecture mooted is supported by facts in its favor, and appears justifiable. Still, even should a communication between the lymphatics and urinary passages be found to exist, the appearance of chyle in the urine will still remain unexplained, since, in the complaint we are discussing, it is not lymph with which we have to deal but true chyle, as the enormous amount of fat which it contains testifies. So we are further forced to the conclusion that, owing to some insufficiency of the valves in the lymphatics, actual chyle from the chyle-duct (cisterna chyli) forces its passage backwards into the lymphatic fistula.

---

[1] *Kühne*, Lehrbuch der physiologischen Chemie, S. 543.

[2] *Heschl*, Wiener medicinische Wochenschrift, 1866, No. 31.

The addition of albumen to urine, made at some part beyond or outside the kidneys, is quite a subordinate matter to the subject which occupies us principally in the present work, and really only merits mentioning for its value in settling difficult questions of diagnosis. We have to deal only, at the present time, with albuminuria renalis—that is, the mixing of albumen with urine in the organs where the latter is being secreted and at the moment of its secretion.

In the course of the last century various practitioners had observed that persons convalescing from scarlet fever often passed bloody urine and were dropsical; and subsequently Cotugno[1] was the first who, in Anno Domini 1770, published his observations showing that dropsical persons could pass urine which, although not blood-colored, might coagulate just like white of egg on being heated. As Cotugno found a substance which gave the same reaction in the dropsical effusions which filled the different serous sacs of the body, he regarded the excretion of albuminous urine in dropsy as only a spontaneous or natural effort of the healing powers, and assumed that this was nature's mode of ridding herself of the difficulty by removing the pathological product through the kidneys. Cotugno's discovery was not lost to the world; it received the consideration it deserved, and was principally established by the labors of some English physicians. Cruikshank took the presence or absence of albumen in the urine of dropsical subjects as his guiding principle for distinguishing one form of dropsy from another. Wells had already noticed, in his pathological inquiries, certain anatomical alterations, to wit, thickening of the cortical layer from deposition of coagulate lymph, in particular kidneys which had secreted albuminous urine during life; but he did not imagine that a kidney must, in every instance, be diseased if it furnished a urine much mixed with serum. Wells, too, was the first person to show the presence of albumen in the urine of a person who was not dropsical and had the appearance of health.

---

[1] *Dominici Cotunnii* de Ischiade nervosa Commentarius. Viennæ, 1770, p. 28.

The frequency of kidney disease in dropsical subjects, and such as had exhibited albumen in their urines, did not escape Blackall;[1] this author, however, maintained that the changes exhibited in the kidney were not the cause of the albuminuria, but were themselves common results of one and the same primary affection, namely, a state of general inflammation; and to this he attributed both the passage of albumen from the blood into the urine and the kidney alterations.

The doctrine of albuminuria entered upon a fresh phase when Richard Bright,[2] in 1827, published his reports of medical cases. Bright showed that certain alterations in the kidney, which had been first accurately described by him, were associated with the excretion of albuminous urine, and inferred that this albuminuria might be accepted as the sign of those pathological changes of the kidney which were besides likely to induce general dropsy. Bright certainly guarded himself, later on, against the objection urged against him, that he had accepted albuminuria as the infallible indicant of one particular diseased condition of the kidneys, or had identified albuminuria with kidney disease; but his actual expressions were so worded that no one could object to the meaning forced upon them, that he really accepted the presence of albumen in the urine as evidence of renal disease, at least in commencement, if not confirmed. Now this complaint, by Bright's account, began in some functional disturbances, but, by persistence in its functional disorder, the structural changes of the kidney would surely follow. The researches and observations both of Christison and of Gregory certainly confirm Bright's doctrine, although objections to its correctness were not wanting;

---

[1] *Blackall*, Observations on the Nature and Cure of Dropsies; and particularly on the Coagulable Part of the Blood in the Urine, etc. Philadelphia, 1820.

[2] Reports of Medical Cases, selected with a view of illustrating the Symptoms and Cure of Diseases by a reference to Morbid Anatomy. London, 1827-31.

thus Graves, Prout, and other writers in Great Britain, asserted that the real cause of the albuminuria and also of the kidney disease was not the alteration in structure of the organs that secreted the urine, but subsisted in some abnornal composition of the blood or in some perverse construction of the albuminous element of blood serum.

These different views are not reconciled even at the present time. While many physicians to-day read the term albuminuria as an equivalent for kidney disease, there are others who attribute some, if not all, such cases to an altered condition of the blood.

Stokvis,[1] of Amsterdam, in his work, entitled Experimental Inquiries into the Pathological Conditions which lead to the Development of Albuminuria, has furnished an important contribution to the etiology of this symptom. The conclusions to which his experiments and his observations forced him, and which he succinctly states at the end of his work, were, that he could not discover any chemical change in the blood to explain the albuminuria, but was forced to attribute this symptom in every case to some disturbance of the circulation and to an increase of the normal blood pressure in the excretory vessels of the kidneys.

If from this brief historical introduction we turn our attention to the subject of albuminuria more minutely, we may set the following conclusion as certainly arrived at:

The combination of albumen with urine is in every case evidence of pathological conditions.

The vessels charged with the duty of secreting the urine, and notably of course the Malpighian bodies, under their normal conditions do not allow of any transudation of the albumen of the blood serum. The fact is one which we may accept as fully established, notwithstanding the opposite opinion expressed by von Wittich, and previously noticed by us, the grounds for its entertainment having been disproved by Stokvis. It is not in harmony with our experience of the process of secretion in other glands, and least of all with the laws of diffusion that obtain outside the body, that albumen can be forced through animal or other membranes except as the result of inordinately high pressure.[1] Without doubt we must take into consideration the epithelial layers covering the capillary tufts interposed within the glomeruli, which subject the fluid within the vessels to a process of spontaneous filtration and serve very essentially to retain the albumen of the serum in the blood.

---

[1] Recherches expérimentales sur les conditions pathogéniques de l'albuminurie. Journal de Médecine de Bruxelles. Vols. 44, 45. 1867.

No departure from a normal condition of the blood can lead to any infraction of the law above stated. The opinion not long since entertained, that mere thinning of the blood-serum (hydræmia) was of itself sufficient to allow overflow of albumen into the urine, has been overthrown by the experiments of Stokvis, which showed that this could not take place when all unnatural increase of pressure in the general circulation was excluded.

The same inquirer admits the possibility of producing such a thinning of the blood, hydræmia, by injecting water into the veins of an animal (after this has been bled beforehand to a corresponding amount, to avoid any unnatural increase of blood pressure), as to lead to a dissolution of the blood corpuscles in the now impoverished serum, thus permitting hæmoglobin, the coloring constituent of the blood-cells, to pass over into the urine. A urine, however, that contains hæmoglobin is blood-colored and gives the reaction of albumen, an albuminous matter being formed out of hæmoglobin, as is shown by response to the ordinary tests for albumen.[2] Stokvis contends for the impossibility of the serum in man ever being so thin by disease as to admit of the solution of the blood-cells, although he was able to effect this artificially in animals. But to the excretion of hæmoglobin by the kidneys I shall have occasion to return hereafter. Wundt thought he had discovered a cause of albuminuria in a lack of common salt in the blood serum. This is denied by Stokvis, upon the grounds of a series of experiments made partly upon himself, when he abstained as far as possible from taking salt, and partly upon animals, which he kept on a diet absolutely devoid of any salt at all.

---

[1] *Kühne*, Lehrbuch der physiologischen Chemie, S. 542.

[2] *Kühne*, l. c. S. 539.

Canstatt, and Prout, too, especially, had asserted that a chemical alteration of the albumen contained in the blood-serum, some error of formation in this, was the true cause of every case of albuminuria ; this alteration being of such a nature that the modified or damaged albumen may be removed by the blood-vessels of the kidneys—each of these writers being in this respect in accord with the other. Neither of the advocates of this opinion, however, was in a position to show that the albumen thus excreted by the kidneys in albuminuria differed in any respect from that contained in ordinary serum. Stokvis injected the albumen excreted with the urine of persons suffering with kidney disease into the veins of animals, without obtaining a trace of albumen in the urine of the animal, and concluded, therefore, that the theory of the existence of some peculiar modification of the serum-albumen, which the kidneys were able to remove from the blood, had no foundation in fact. On the other hand, his experiments entirely confirmed the observations made by Berzelius long before him, that raw white of egg, injected either into the veins or subcutaneous cellular tissue of animals, quickly makes its way out into the urine. He pointed out the difference between the albumen of the serum and that of white of egg, appointed by the different conduct of the two substances when treated with nitric acid ; both are precipitated from their solutions by this acid, but the precipitate of serum-albumen is easily and completely redissolved in an excess of the acid, while that of white of egg albumen dissolves with extreme difficulty, and never completely. White of egg albumen, taken raw and in large quantities as a sole article of food, passes over into the urine, according to the observations of Tegard, Brown-Séquard, Hammond, and Claude Bernard, who experimented with it. Stokvis, after feeding himself and his friends upon a very large amount of raw eggs, was unable to detect any albuminuria in consequence of it ; still rabbits thus exclusively fed by him, as a rule, excreted albumen in their urine ; the albuminuria, however, even then was arrested when he gave them cooked or coagulated instead of raw white of egg.

Stokvis upon this assumed that raw white of egg could be absorbed directly and unchanged from the stomach, and be excreted again at once by the kidneys, and that coagulated albumen could only reach the blood-vessels after previous digestion and conversion into peptone. He further disputed the fact, avowed by some writers, of the presence of peptone in normal urine. Serum albumen, given by mouth to rabbits, produced in his hands no albuminuria.

Apart, then, from the possibility of raw white of egg being directly absorbed into the circulation, and the passage of hæmoglobin into the serum as a decomposition product, there is, according to Stokvis, no such thing as an albuminuria of pure blood origin having its sole source in some abnormal state of the blood serum.

In his experiments, however, Stokvis evidently overlooks, or rejects with unfairness, the possibility of other albuminous substances besides serum albumen, of which not a few are contained in the blood, passing over into the urine. Since J. C. Lehmann,[1] of Copenhagen, first stated with positiveness that every specimen of albuminous urine contains, besides serum albumen, *globulin*, the fact has obtained the fullest confirmation, and this most recently at the hands of Edlefsen[2] and Senator.[3] The circumstance itself ought to surprise us less, because globulin,[4] unlike ordinary serum albumen, can diffuse itself through animal membranes—nay, rather it ought to astonish us that this substance, which is always contained in normal serum, should not, by reason of its property of diffusibility which we have learnt, pass over constantly into the urine of healthy persons.

Lastly, Gerhardt has made some observations,[5] showing that an albuminous substance, precipitable neither by boiling nor by nitric acid, but easily detected by alcohol, may be present in the urine. This condition of things he denominates latent urine albumen, and seeks to explain its latency either upon the ground of some peculiarity in the constitution of the albumen itself, or upon the presence of some peculiar elements in the urine which interfere with the production of the ordinary albumen reaction. In a later communication Gerhardt gives another explanation of this anomaly, stating that it is due to a decomposition of serum albumen into peptone under the influence of a fever temperature, just such as may be accomplished in the laboratory by protracted boiling (Wiener med. Presse, XII. Jahrgang, 1871, 1). Senator asserts that peptone appears in small quantity in every urine that contains albumen, and supports his statement by several observations and tests.

---

[1] Zur Chemie des Eiweissharns. Virchow's Archiv. Bd. 56, S. 125.

[2] Beiträge zur Kenntniss der Eiweissstoffe des Harnes. Deutsches Archiv f. klin. Med. Bd. 7, S. 67.

[3] Ueber die im Harne vorkommenden Eiweisskörper u. s. w. Virchow's Archiv. Bd. 60, S. 476.

[4] *Kühne*, l. c. S. 223.

[5] Ueber die Eiweissstoffe des Harnes. Deutsches Archiv für klin. Med. Bd. 5, S. 212.

This last-mentioned fact serves, however, in no wise to support the doctrine of an albuminuria of blood origin in the sense intended by Canstatt and Prout and their followers.

In every case the explanation of the excretion of albumen by the kidneys must be sought for in disturbance of the processes by which the secretion of urine itself is effected.

It follows that the next point to which our attention must be directed is the anatomy of the kidney itself, and what changes they are that take place in the tissues that compose these organs; and first and foremost we must speak of *the cells that line the renal tubes.* Careful examination has taught others as well as myself that it is no such rare event for kidneys to secrete albuminous urine during life, which yet after death present no trace of histological alterations, either in their (glandular) cells or in their interstitial tissues; while, on the other hand, kidneys which are extensively diseased, whose gland-cells and interstitial frameworks exhibit pathological degeneration of extremest kind, may furnish a urine totally free from albumen.

At all events, then, pathological alterations of the epithelium, or of the matrix tissues of the kidneys, must be of very secondary importance in the production of albuminuria. At the same time Senator is perfectly right in saying that through loss of renal epithelium, and in consequence of disturbances of nutrition of most various kinds, albumen or some modification of it—myosin—may escape from the nutrient fluids and be mixed with the urine. But, surely, the quantity of albumen thence arising would be a very minimum.

The overflow (passage) of serum albumen from the blood-vessels into the renal tubes of the kidneys will in every instance be proportioned either to an existing abnormal increase of the blood-pressure, or to an altered state of the walls of the vessels, or to a combination of both these causes acting together.

I shall endeavor to show hereafter the correctness of this proposition, and to apply to important clinical cases of albuminuria this theory, which accords generally with the conclusions arrived at by Stokvis, in his inquiries into the conditions requisite to produce albuminuria.

Experience teaches us that animal membranes, such as are employed for purposes of filtration, are so little permeated by albuminous substances, even when filtered under very high pressure, that a very small percentage amount of albumen passes through the filter; still, the albumen content of the fluid thus being filtered remaining the same, we perceive that the albumen contained in the filtrate increases directly in quantity according to the degree of pressure employed. Now, this may happen, because the filter membrane or medium becomes more stretched under the greater strain, and because its pores are correspondingly enlarged.

The vessels of the kidney from which the urine is secreted, the capillary coils of the Malpighian tufts, are the parts which we must regard as filters of the above-mentioned kind, which, under normal circumstances, as above noticed, never allow any transudation of the albumen of the blood serum. The permeability of this filter, however, like that of the animal membrane employed in experiments outside the body of an animal, increases with the pressure employed; and then it may chance that the cells which form the covering of the capillary coils loosen and separate from each other under the strain to which the glomerulus is subjected, and admit of pores, or passages, forming in the epithelial network; thus it becomes conceivable how the coats of the blood-vessels of the kidney may come to allow the transudation of the serum albumen through their pores, without suffering further change of structure, directly the blood pressure bearing upon them exceeds certain bounds.

Thus perfectly healthy kidneys, solely in consequence of an *altered state of blood pressure upon their vessels, can excrete albuminous urine.*

This fact has been further confirmed by experiments upon animals, by stopping the return of blood through the renal vein. On the other hand, recent experimenters and very careful operators, who have artificially increased the pressure in the arterial system, have altogether failed thereby to induce albuminuria. Later on, however, I shall be able to prove that abnormal pressure in the arterial renal vessels is the sole cause of albuminuria in particular pathological conditions.

Albuminuria may arise, as an entirely transitory symptom, from abnormal elevation of the blood pressure, and pass off again as quickly as the cause from which it arose; but, on the other hand, this cause abiding, it, too, may endure; and lastly, it may come and go, fluctuating with the varying grades of pressure which produced it. It is a constant rule, however, that when albuminuria exists, which is the result merely of undue pressure upon the vessels of otherwise sound kidneys, and is not dependent upon alterations in the coats of these vessels, the actual percentage amount of albumen held in the urine is but little; and the results are the same as those which we observe in the filtration of albuminous fluids, under pressure outside the body, when the actual quantity of albumen which transudes under any great pressure is perfectly trivial compared with the amount contained in the fluid thus employed.

In the clinical cases of *albuminuria due to heart disease*, the process which takes place agrees most closely with that which is instituted artificially when we increase the blood pressure in the renal veins. Persons who have serious valvular obstruction, especially those who have mitral stenosis, and those who are affected with extensive muscular degeneration of the heart, excrete urine, which constantly fluctuates in its albumen content, being now albuminous, now free from albumen, according as the cardiac lesion is little or severely oppressing the general circulation of the blood. Thus so long as care and treatment are able to obviate or diminish the disordered circulation, a diminution of the albuminuria can be confidently expected, as temporary or enduring evidence of benefit received. In this class of cases the passage of albumen into the urine is dependent upon structural alterations of the kidneys, which we perceive, in fact, in the dead body to be quite indistinguishable from what is recognized as cyanotic induration, and which is due to varying degrees of pressure compelled by disturbance of the general circulation.

For instance, the urine of these patients contains albumen directly the pulse at the wrist falls to its lowest ebb of weakness and the stasis of circulation in the veins advances to established cyanosis, and then, in consequence of the diminished arterial pressure, the quantity of urine excreted per diem falls to a minimum and its specific gravity rises above normal, until at last hydræmia and dropsy ensue, on account of the diminution of the renal secretion and the retention of water in the system associated therewith. Again, in these patients, the albumen disappears from the urine, as soon as the arterial pulse becomes stronger and the cyanosis less marked, and as soon as larger quantities of a more watery urine are excreted. The urine becomes permanently albuminous, when the general symptoms of cardiac inadequateness above described admit of no abatement, because they are no longer temporary.

Entirely agreed as all authors are in their description of this form of albuminuria, it still appears to me that here and there somewhat obscure ideas are entertained upon its immediate mechanical cause. Thus it is assumed that a congestion of the renal veins takes place and extends backwards through the capillaries to the vasa efferentia, reaching to and distending the Malpighian tufts, that an increased pressure is thus produced within the coils of the glomeruli, until at last albumen becomes squeezed through the walls of the capillaries. Now a venous congestion, reaching up to the tufts themselves, as a consequence of disturbance of the circulation, such as we have here under discussion, would be rare and exceptional indeed, since "the vasa efferentia," as Senator[1] rightly enough remarks, "present a more favorable mechanism for obviating local and general stasis than we discover in any other capillary apparatus of the whole body, splitting up, as they do, into a duplex capillarization, two sets of networks really intervening between the artery and the vein. Any obstruction to the venous outflow will press first and hardest upon the interstitial capillary system, and thence be directed backwards, in the direction of least resistance, upon the renal arteries. While, therefore, the interstitial capillaries, like those of the rest of the body, will experience the full impact of an augmented venous tension, the capillary coils inside the glomeruli themselves can have only a part of this additional burden to support." [1]

---

[1] L. c. S. 496.

Now since the pathological conditions which bring about such a general stasis of blood in the venous system of the kidneys are necessarily accompanied by a diminution of arterial pressure, the vessels of the Malpighian bodies, in which variations of arterial pressure are principally felt, must experience a less than normal strain. "For in them the diminution of arterial pressure is felt in its full entirety, the increased venous tension only in some degree" (Senator). After death one finds the glomeruli of the cyanotic indurated kidney only moderately filled out—indeed, according to some authors, they are in part atrophic. Klebs is of opinion that even slight increase of pressure in the arterial system is sufficient, when the venous outflow is rendered at all difficult, to lead to the escape of albumen or blood from the glomeruli. This opinion is certainly completely correct of those rarer cases in which the venous outflow is obstructed by obstacles of a mere local kind, as, for instance, by thrombosis of the renal veins, the arterial blood-stream continuing to reach the kidneys at its normal rate of pressure. I had the opportunity of observing a case of this kind in the year 1865.[1]

---

[1] TRANSLATOR'S NOTE.—The above quotation from Senator scarcely states the peculiarity of the blood capillarizations so clearly as to enable the reader to master the exact situation of affairs and realize its indisputable importance. The glomerulus truly hangs, like a currant, on its efferent arterial stalk, and the first capillarization takes place in its interior; the issuing efferent vessel, the trunk of reunion, next breaks up into the second network, which spreads between the curling or secreting tubes; far the larger quantity, if not quite all the blood thence resulting reunites into main trunks, the so-called arteriolæ rectæ (vasa recta, Donders), which, thirdly, and lastly, split up into capillaries, which course beside the straight or excretory medullary renal tubes, and finally unite to form the radicles of the renal veins.—(For further details, vide paper by translator in Vol. I., p. 176, of St. Bartholomew's Reports, London, 1865, entitled "Minute Structure of the Kidney.")

CASE I. A robust, well-made man, forty-four years of age, was brought to my Clinic in June, 1865, on account of an enormous œdematous swelling of the lower half of his body, which reached some little distance above the vault of the ribs into the soft tissues covering the thorax. The œdema was distinguishable from that of ordinary anasarca of the subcutaneous tissues met with in kidney disease, because of the deep cyanotic aspect of the swollen skin, the smallest superficial veins appearing blue-red upon its surface, and being scattered about in all directions, enormously distended, giving an appearance which contrasted in a very remarkable manner with the extreme pallor of his face. The enormous enlargement of the principal cutaneous veins, which formed thick networks on both sides of the body, in the lumbar region, and over the anterior abdominal wall, further appointed a causation quite distinct from renal disease. This enlargement of the cutaneous veins extended anteriorly up to the lower intercostal spaces where the thick trunks penetrated into the deeper structures. The heart and lungs presented nothing abnormal about them, and the radial pulse, beyond being moderately accelerated, was not anomalous. The peritoneal cavity contained no fluid, although the abdominal walls were, as above noticed, so extensively swollen and œdematous. The patient passed a fair quantity of urine (1,640 c. ctm. daily), a urine, as a rule, manifestly tinged with blood, and whose specific gravity ranged from 1011 to 1013; it contained albumen, in pretty large quantity, constantly, and presented a sediment of red blood-cells, epithelial cells, and casts.

The diagnosis made was thrombosis of the vena cava ascendens, although it was impossible to explain the thrombosis, the patient being unable to tell us anything that threw any light upon its causation. On the 17th of August he died, a large quantity of fluid having collected in the peritoneal cavity and in the right pleural sac before his death.

At the post-mortem the liver was found firmly attached to the diaphragm and other neighboring organs by thick bands of adhesion. Hazel-nut sized gummata were scattered throughout the liver substance; the vena cava ascendens was tightly compressed at the point where it enters into the groove at the posterior border of the liver; its wall was wrinkled lengthways and plugged with a very old clot, which was in a state of fatty degeneration. This thrombus could be tracked downwards and peripherally through both venæ iliacæ into the crural veins. The tunica adventitia of the vena cava was thickened at the spot where this vessel enters the liver groove, and the liver substance in its immediate neighborhood was entirely converted into a callous connective tissue; this it was that had effected the narrowing of the sulcus in which the vena cava lay. Except in the liver, there was no trace of any other syphilitic lesion in the body.

---

[1] The case is fully described in an Inaugural Thesis by *Dr. G. Dreis.* Venarum thrombosis tria specimina. Kiliæ, 1866.

This case shows how the urinary excretion conducts itself when the current from the renal veins is obstructed, while the arterial blood-supply is unaffected. The daily quantity of urine remains abundant, but its specific gravity is below normal—while albumen, and generally blood too, is present in the secretion. Quite otherwise are the conditions under which secretion is conducted when there is general venous stasis, and the urine is albuminous in consequence of cardiac incompetency; then in the degree in which the pulse becomes small and feeble, the daily quantity of urine secreted diminishes, falling to less than a hundred c. ctm., while its specific gravity rises to 1035 or even 1040. Albumen and traces of blood are then discovered for the first time in the patient's urine, when his pulse has become scarcely perceptible and the blood tension in his arterial system has sunk to its lowest ebb. The albuminuria in such cases sometimes disappears with surprising rapidity directly the pulse recovers itself, the increase of arterial pressure procuring the excretion of a larger quantity of urine of lower specific gravity. Now, why have we albumen in the urine in these cases? Cohnheim's researches upon venous congestion give us a precise answer to the question. Cohnheim has shown that, following upon stasis in the veins, we have not merely escape of blood plasm, but actual emigration of red blood-cells through the walls of the capillaries into the surrounding tissues belonging to that particular province of which the veins are obstructed. I am altogether of the opinion entertained by Senator, and which I have previously expounded, that the relatively scanty albumen and blood, which the urine contains in cases of heart disease with cyanosis, finds its way into this secretion, only after it has reached the renal tubules, and emanates from the capillary network surrounding these.

A striking proof of albuminuria solely due to alteration in the blood pressure, and at the same time of the dependence of this symptom upon the pressure alone, is furnished in the human subject by the following remarkable case of illness:

CASE II.—A tall, shot-up lad, of sixteen years (H. from N.), had been suffering for five years with an illness attended by fever, which had kept him a long while confined to his bed. His father called his illness nervous fever (it was probably myelitis). In consequence of it, extensive atrophy had set in over several groups of muscles, while certain other sets had become stoutly developed. Thus the muscles of the upper parts of both his arms were but sorry relics, while those of his forearms remained unusually powerful. The pectorales had wasted away nearly altogether, and the abdominal muscles were of much diminished volume. The muscles which nod the head, the sterno-cleido-mastoids, on both sides were hypertrophied, but the M. cucullaris (trapezius) of the right side was very feeble, while that of the left side was very powerful; the glutæi on both sides were soft and feeble; the longissimi dorsi were well developed; the muscles of both lower extremities were wasted, those of the right more than those of the left, and most especially at the lower part of the thigh on its anterior aspect. As a consequence of this, so uncommon and so extensive a muscular atrophy, it followed, amongst other things, that the pelvis had become bent over from above downwards to a most remarkable extent, and that the young fellow, to be able to stand upright and get about at all, had been compelled to stretch his spine so that this appeared bowed out in front in the thoracic portion (lordosis). Directly the patient sat or laid himself down, the natural curve belonging to the spine in its thoracic part, its convexity towards the back, reappeared. But this condition of things had further led to the actual diameter of his thorax, from behind forwards, being rendered considerably less when he got up and walked than when he sat or lay down—so much so that the diameter of his chest measured, from the root of the ensiform cartilage to the spinous process of the ninth dorsal vertebra, 13½ ctm. when he stood up, as against 18 ctm. when he was sitting, and a full 16 ctm. when he lay horizontally upon his back.

If we reckon now the following measurements as to the thickness of the ninth dorsal vertebra, estimated through its spinous process, or inclusive of this, at 7 ctm., which was what I found it to measure upon the skeleton of a slightly built person, and allow 1 ctm. for the thickness of the sternum, and then deduct the sum of this from the measurements of the chest of this lad, we shall find that there remains at most a space of only 5½ ctm. between the backbone and sternum, at the level of the base of the ensiform cartilage, when he stood upright, and even less than this at a diameter taken at a slightly higher level. Now, when he stood upright his heart was driven forwards against the anterior chest wall, in a remarkable and quite unmistakable manner.

The shape of the cardiac area of dulness when he stood up exceeded the limits marked out when he was lying down, both upwards and to right and left, more than a centimetre, and the third, fourth, and fifth left ribs were plainly pushed outwards and forwards at each systole of the heart. In consequence of this compression, the contractions of the heart took place with far greater rapidity when he was in the standing than when he was in the lying posture. Thus when he was recumbent his pulse numbered from seventy to eighty beats per minute; but it rose to from one hundred to one hundred and twenty directly he stood up; and lastly, so long as the boy was in the erect position, one heard a loud systolic murmur over the lower part of the sternum, although at the apex beat and at the point of origin of the two great arteries the heart sounds were quite clear. If, after such an examination, undertaken while the boy was standing, he was allowed to lie down, it was found that the pulse fell instantly to seventy-two beats, the ribs were no longer elevated at the systole as before, and the systolic murmur disappeared at once from over the lower part of the sternum, leaving the heart sounds sharp and clear. The explanation I assigned to the above remarkable symptoms was as follows: by reason of the abnormal incurvature of the thoracic portion of the spine, the deep diameter of the chest was so shortened that that portion of the heart which lay between spine and breast-bone became compressed. Now, the portion of heart which fell in this situation was the right ostium venosum; this orifice, therefore, must suffer notable alteration of shape under the condition of things which existed, since the flexible ring of insertion belonging to the tricuspid valve must needs accommodate itself to the pressure made upon it. In lieu of the ostium venosum being a circle, it would then become an oblong or some such shape, while the three valve lappets, albeit structurally normal, would be unable to close the orifice; thus there would ensue a relative incompetency of the valves when the lad stood up, although directly he lay down or sat down, and the heart was allowed to fall into its natural posture, and its right ostium venosum to assume its ordinary shape, its valves would close again completely.

Hence I attributed the systolic murmur to a relative insufficiency of the tricuspid valve. The same circumstance, too, explained, I thought, a further remarkable symptom which he exhibited. On the days during which he went about most he excreted albuminous urine regularly, whereas the urine he passed by night was equally, as a rule, entirely free from albumen; and upon the days when I kept him in bed his day's urine, too, presented no albumen. Now, in only one specimen of the urine he passed by day did I find a cast, and this was a perfectly hyaline one.

Now, the conduct of the urinary secretion under the conditions above stated is so persistent, it has been proved so carefully and under such repeated variation of the circumstantia and modificantia, with such invariably identical results, that any error of observation may be confidently set aside.

Then, if the cardiac explanation of the symptoms which I have adduced be the correct one—and, if it be not, I cannot tell what interpretation to set up in its stead—when the lad is upright, at each systole of his heart a blood wave is directed from the right ventricle backwards and upwards into the right auricle, and thence into his general venous system, and the blood pressure upon his veins must be enormously increased by day when he is about on his legs; and in this way, I apprehend, an overflow of albumen of blood-serum takes place at those interstitial capillaries which are placed round about the tubules carrying the urine out of the kidneys (the excretory tubes); and so long as the venous pressure obtains, just such an outflow of albumen takes place as is the abiding consequence in cases of grave cardiac incompetency.

Dr. Max Huppert observed transitory albuminuria after epileptic attacks (Albuminurie ein Symptom des epileptischen

Anfalls. Virchow's Archiv. Bd. 59, S. 367), in which, too, the albumen was far more copious after a severe than after a slight or abortive fit. Dr. Huppert does not attribute the epileptic albuminuria to the violent convulsive phenomena of the seizure, since the same albuminous urine, although in less degree, follows upon the so-called epileptic vertigo, in which no convulsion whatever occurs. For the rest, it is self-evident that, beyond valvular and other lesions of the heart, anything which disturbs the circulation and so entails similar effects, namely, considerable venous congestion, will produce albuminuria. To this category of affections belong pleuritic effusions, obliteration of several branches of the pulmonary artery, such as follows upon cirrhotic degeneration of the lung substance, or is the consequence of pulmonary emphysema. Still, as a rule, the above affections of the respiratory organs do not often lead to congestive albuminuria. Nor must we confound with them the albuminuria of phthisis ; for in phthisis, as is generally known, well-marked cyanosis is quite exceptional, and this notwithstanding so large a portion of the domain supplied by the pulmonary artery has been destroyed ; moreover the hectic fever which accompanies the disease so quickly diminishes the general mass of the blood that the blood-vessels still remaining patent offer room enough to carry on the circulation, and hence no backward pressure takes place, and no congestion of the general system remains. But yet phthisical subjects often do excrete albuminous urine ; this is so, but it is for other reasons, which I shall have to speak of hereafter. Lastly, there is still another form of transitory albuminuria which we must consider—rare enough in itself, but also dependent upon changes in the blood-pressure, although these are merely local and confined to the renal vessels. I have only had the opportunity of observing one example of this myself—I mean of albuminuria which arises after long and persistent blocking up of the ureters. I have already made mention of this case in another work.[1]

Case III.—R., inspector of economics, aged 28. robust, had already experienced several attacks of renal colic when he came under my care, on December 15, 1867.

---

[1] Sammlung klinischer Vorträge. Red. v. R. Volkmann. No. 25.

On the day after his arrival here in Kiel, he was seized with a severe attack o colicky pains, and this time in both kidney regions. Together with the fearful pai he was suffering, he had a constant sensation as if he were choking, continuous vomiting, and an almost incessant though fruitless desire to pass his water. Three day later his pain left him, but the vomiting continued; for one hundred and twenty two hours he had not passed one drop of urine, and the bladder, which was explore by a catheter, was quite empty. Then, with subsidence of all his symptoms, ther ensued such a copious discharge of water, that in twenty-four hours he had excrete 3,025 c. ctm. of urine. The urine had a specific gravity of 1009, and contained albumen, and in its sediment, besides red blood-cells and epithelium, from the pelvis o the kidney and the ureter, there was a quantity of quite hyaline, but for the most par very thick casts, although some smaller ones accompanied them in which wer clustered the ordinary well conditioned epithelial cells from the renal tubes. Upo the re-establishment of the urinary secretion, he felt completely well again, and hi appetite returned; but for four days the water, still copiously secreted, continued t be albuminous, although so early as on the third day no further casts could be discovered. On the second day after the urine flow had returned, I found a fairl large quantity in the sediment, but noticed that instead of their being hyaline, a observed upon the first day, they were clouded by numbers of tiny particles o highly refractive fat drops. Fortunately (for my own re-assurance), the urine o the previous day was at hand, in which the casts, all of them, still maintained thei perfect hyaline appearance. It was not till six weeks after this serious attack tha our patient passed a calculus about the size of a bean.

According to Max Herrmann's researches[1] into the effects o pressure upon the glomeruli and urinary tubules, we are led t interpret the albuminous urine occurring in this case in the following way: the passage of urine from the pelvis of the kidney being obstructed, a distention of the latter at once take place, and of necessity there ensues a blocking up of the urin tubules themselves. "Under such conditions all the blood-vessels must be subjected to pressure, and the veins certainly wil suffer greater compression than the arteries, so that the bloo will flow less quickly through them, circulating, as perforce i must, through the kidney under conditions of greater than normal tension." This view of Herrmann's is supported by the following experiment which he made: "A dog was narcotized, an a canula placed in its renal vein to intercept the blood flowin through it. The ureter was now filled with water, and this wate was alternately submitted to and relieved from the pressure of thirty-five mm. of mercury; each time the ureter was thus distended, the blood current through the renal vein, at all times pretty rapid, became slackened, and was notably less rapid than when pressure upon the side of the ureter was removed." If we apply Herrmann's theory to the explanation of our case, we shall be led to the conclusion that, as the result of long-continued suppression of urine and consequent increase of tension in the arteries, the capillary coils of the glomeruli become greatly distended and stretched beyond their normal calibres; and then when the pressure is removed, and free outflow of the urine once more established, the walls of the capillaries, still remaining stretched for some little time (being unable to resume their normal size at once), allow the albumen of the blood serum to transude.

---

[1] Zeitschrift für rationel. Medicin. Dritte Reihe. Bd. 17, S. 25.

Similarly, as an entirely temporary symptom, and then, to be sure, one which does not depend upon any structural change in the kidneys, we meet with *albuminuria* occurring exceedingly frequently *in persons who are suffering from some severe febrile movement.* In these cases it is due to the extremely high elevation of the temperature of the body, and to its long maintenance, be the cause of this fever heat what it may. This febrile albuminuria occurs in the course of severe angina, in pneumonia, in typhoid fever, and in the congestive stage of the acute exanthemata, in pyæmia, etc. As the fever declines, and directly in the more acute febrile diseases that defervescence begins, the albumen, usually only sparing in quantity, disappears from the urine as suddenly as it came; but should the fever terminate fatally, when the kidneys are examined at the autopsy no pathological changes, such as explain the albuminuria which most certainly obtained during life, will be discovered.

The pathological state designated as "cloudy swelling," so often described in the kidneys of persons dying of severe febrile disease, cannot be the cause of the albuminuria observed during life, for on the one hand we occasionally find kidneys presenting this aspect, which come from persons who during life never presented a trace of albumen; and, on the other hand, we sometimes discover no cloudy swelling when albuminuria was present during the fatal illness. Cloudy swelling of the renal epithelium, therefore, may well be, when not entirely a mere post-mortem change, as some suppose it, the co-result of the same active agency as that which produced the albuminuria; but it is not the cause of this.

Gerhardt[1] was the first person to differentiate and distinguish this albuminuria of fever from that which is strictly renal and due to structural changes in the kidney. In the course of his investigations, already alluded to, he arrived at the idea that in renal or kidney-disease albuminuria, it was more the serum albumen that passed into the urine, while when this symptom was merely incident to the febrile state, the albumen substance represented the rapid disintegration of a great number of the red blood-cells. Gerhardt, however, was unable to point out any very obvious difference between the albuminous material furnished in these differently originated forms of albuminuria; although, in some cases of the febrile albuminuria, this substance appeared in what he calls the latent or peptone form, when it is not precipitable by boiling after addition of nitric acid. But then his so-called latent passes into the ordinary form of albumen after a matter of one or two days.

Obermüller[2] adduces further similar observations. As for myself, I have not had time to undertake any extensive examination of the urine in fevers with the view of ascertaining its peptone content; years since I considered the question of ordinary albumen in urine of this kind, and then I could not make out that that which was present in febrile albuminuria—using this term in its ordinary acceptation—differed at all, in its response to the usual chemical agents, from what is furnished in persistent albuminuria. One must therefore agree with Gerhardt, when he says that in the subjects of acute fever, whose temperature stands persistently above 40° Cent. (104° F.), albumen passes into the urine, because the process of filtration, by which the urinary secretion is effected at the Malpighian bodies, is, by reason of the abnormally elevated temperature, conducted under entirely abnormal conditions.

---

[1] l. c. S. 213.

[2] Beiträge zur Chemie des Eiweissharns. Inauguraldissertation. Würzburg, 1873.

We are now led to inquire what influence this elevation of temperature can have upon the product of filtration, and how this is affected by it; and the first thing we perceive is, that elevated temperatures relax the walls of the blood-vessels, or make them yield more than they otherwise would to the hydrostatic pressure of the blood streaming through them.

This febrile albuminuria, then, is most akin to the overflow of albumen into the urine, which follows when the vaso-motor nerves of the kidney are divided. It may be well to dismiss the question, whether this relaxation of the walls of the vessels follows because of some perversion of the nerve influence governing the muscular elements of the arteries, consequent upon the high temperature, or whether it is the elastic element that thus suffers loss of its faculty of resistance. But in either case, the excessive heat of the body acts exactly in the same way as section of the vaso-motor nerves; it is the nature of the filter by which the urine is secreted that is disarranged. For inasmuch as the surface of the filter is enlarged, its pores must of necessity become more roomy. In my own opinion, therefore, we need no such elucidation of febrile albuminuria as that which has been furnished to us, involving as it does assumptions which have no facts to justify them, to wit, that in consequence of a more rapid destruction of red blood-cells, not their own decomposition products, but some other out-of-the-way albuminous substance, finds its passage into the urine, a substance for which the walls of the Malpighian coils are more porous than for ordinary serum albumen.

Of course, the result of this alteration in the conditions of filtration at the glomeruli will vary according to the individual; under apparently identical circumstances, we shall in one case have albuminuria, and in another none. It is not every patient whose fever temperature rises above 40° C. (104° F.), who will excrete albuminous urine: in one, albuminuria will occur after an elevation of temperature of only short duration; in another, a fever heat of considerable endurance will be requisite to produce a like result.

Now, however much it is the rule for febrile albuminuria to be quite a transitory matter, an event wholly independent of all pathological change in the kidneys, still I will not deny that, after prolonged continuance of the febrile state, actual disturbance of nutrition in the tissues of the kidney can ensue in certain cases, and may affect in particular the walls of the blood-vessels, pursuing afterwards a chronic course, and eventuating in albuminuria of a permanent kind, just as we find that degenerations of the severest character follow without exception upon sections of the vaso-motor nerves in the kidneys of animals, if only they survive the effects of the operation a sufficiently long time. Still it is certainly extremely rare for an inflammatory kidney disease, with persistent albuminuria, to follow upon typhoid fever, pneumonia,[1] pyæmia, small-pox, or the like. Numerous, too, as have been the cases of febrile albuminuria which have come under my observation as complications of the above-named acute affections, the examples have been only exceptionally few in which the albumen has persisted in the urine after the fever has remitted, and in which dropsy has followed, indicating true disease of the kidneys. My experience tallies altogether with the statements of Buhl, who says that out of three hundred cases of typhoid fever terminating fatally, he only met with general dropsy of kidney origin once or twice.

Let it be understood, then, that I entirely separate the acute diffuse nephritis which so often occurs in diphtheria, relapsing, and scarlet fever, from febrile albuminuria, since in these diseases we have to deal with some specific implication of the kidneys which has nothing whatever to do with the fever heat accompanying or experienced in the course of these complaints.

Stokvis, in his often-cited work on albuminuria, only allows the nerves the following limited influence in the production of albumen in the urine, namely, that paralysis of the vaso-motor nerves may bring about sŏme disturbance of the circulation through the kidneys. He attaches great importance, in this connection, to the discoveries made by von Wittich, which show that by section of the nerve branches, which lie between the renal vein and artery, and which he considers to be the nerves special to secretion, no albuminous urine results; while section of the special vaso-motor branches, distributed around the artery, produced albuminuria in marked degree.

---

[1] Lungenentzündung, Tuberkulose und Schwindsucht. München, 1872, S. 47.

Stokvis further informs us that the causes producing paralysis of the vaso-motor renal nerves are not invariably to be found in the immediate neighborhood of the kidneys, and directs attention to the physiological experiments of Krimes, Schiff, Longet, and Claude Bernard, which bear on this subject and prove that injuries of certain parts of the brain (the medulla oblongata, the peduncles of the cerebrum and cerebellum, etc.) can excite albuminuria. Prof. Fischer,[1] of Breslau, in a lecture on concussion of the brain, describes transitory albuminuria as one of the very common symptoms, although attaching to it a somewhat different explanation from that which we have given. Again, the occurrence of albuminuria in cerebro-spinal meningitis, which is put forward by some writers as a constant symptom of this complaint, admits of explanation in a similar way. At the same time I feel bound to mention that my experience, as well as that of some others, enables me to state that it is not every case of cerebro-spinal meningitis which presents us with albumen in the urine—indeed, it is quite open to dispute if the albuminuria, occasionally observed in this malady, is not most correctly to be attributed to the incidental fever.

In the preceding pages we have discussed those cases only in which albuminuria is merely the accompaniment of other acute disease—the outcome solely of secondary disturbance of the circulation through the kidneys, the albumen coming and disappearing commensurately with this.

As opposed to these, we shall now make mention of a different class of cases, where albumen in the urine is the prominent symptom, owing to the fact that the kidneys of those furnishing it have suffered a notable alteration of structure. It is still, however, the renal blood-vessels which are either directly or indirectly involved in the structural pathological changes, and the symptoms are the result of altered conditions of blood-pressure localized in the kidneys. That some alteration of the epithelial cells lining the renal tubes, can of itself produce albuminuria, is up to the present time at least not demonstrated—nay, I can assert with confidence that conditions exist in which extensive fatty degeneration of these epithelial cells may have taken place without albuminuria being present. Even Rosenstein observed[1] that, in spite of extreme fatty degeneration of the renal epithelium, no albumen might be present in the urine; and this clinical experience accords completely with the results arrived at by Stokvis from his experiments on animals.

---

[1] Sammlung klinischer Vorträge. No. 27.

Speaking generally, then, it is not every disease of the kidneys that gives rise to albuminuria; in the deposition of tubercle in these organs it is for the most part absent. Further, we have renal diseases in which it is certain that only a portion of the secreting apparatus excretes albuminous urine; and in this category we may include all the affections of the kidneys in which groups of deposits take place, the metastatic processes following an embolism, renal abscesses of other origin, the cancerous and other tumors of the kidney, all of which generally give rise to albuminuria. It is self-evident that in these localized affections the albumen of the urine cannot be derived from the local deposits themselves, for just the spots they occupy are either completely destroyed, or, to say the least, have surrendered their functional activity. The lodgment of a new growth in the renal structures, by compressing or blocking up the blood-vessels in its immediate site, can only give rise to collateral blood-flux and congestion of the neighboring vessels. In the parts immediately surrounding such groups of deposit one discovers the blood-vessels therefore generally distended with blood, and the renal tubules sometimes tinted with blood that has escaped into them. But at a little distance off them we may find the renal substance and its vessels perfectly normal. When blood and albumen appear in the urine, as the consequence of renal hemorrhagic infarctions—and that they produce such a result, I am convinced—they disappear again usually very quickly. In carcinoma of the kidney, too, both my own experience and that of others teach me that the urine may remain free from albumen throughout the entire course of the disease.

---

[1] **Die Pathologie und Therapie der Nierenkrankheiten. Zweite Auflage, S. 29.**

The albumen in localized affections of the kidney—apart, of course, from the accident of an abundant hemorrhage—is always scanty, evidently because a small vascular territory only admits of its escape, and because the small increment thus added is mixed with the entire product of secretion furnished from both organs. But even in the diffuse renal diseases, it is chiefly the associated disturbance of circulation through the vessels of the kidney, which, according to the circumstances of the case, produces either transitory or persistent albuminuria.

The albuminuria in all cases of simple contracted kidney (cirrhosis of kidney, granular atrophy) is solely due to increase of blood pressure in the Malpighian bodies. In such cases a larger or smaller portion of the glomeruli, together with their appendages, waste away as the process of contraction advances, and yet we find that the remainder of the secreting vessels, with their corresponding tubules and the epithelium lining these, is in a perfectly normal state. But the pressure of the blood upon the vessels that remain must of necessity exceed what is normal, since these have to receive the full current from a large arterial trunk, of which a number of natural branch channels are closed. The hypertrophy of the left ventricle of the heart, which, as a rule, accompanies the process of contraction in the kidneys, lends additional force to the already increased pressure of the blood in the renal arteries.

The character of the urine excreted by contracted kidneys corresponds exactly with the conditions under which it is secreted. Thus in the same interval of time, albeit the secreting surface is diminished, an unwontedly large quantity of urine is separated ; for the filtration through the glomeruli being conducted under an accelerated blood current, and under increased pressure, the rapidity of secretion is proportioned to both these. The fluid, filtered through Bowman's capsules under pressure at its back, drives that which is before it through the renal tubules into the excretory channels in a shorter than ordinary period, so that there is less time left for any material alteration of the original filtrate to take place by means of diffusion and absorption into itself of the specific elements of the urine, as furnished by the renal epithelium.

The watery urine of these patients contains, as a rule, small quantities of albumen, and is indeed a filtrate of albuminous serum, driven under abnormally high pressure through the membraneous walls of the capillaries in the glomeruli. Such urine very rarely contains one-half per cent. of albumen, at times hardly one-half per thousand, and in fact there are cases of advanced contracted kidneys which furnish urine some portions of which only now and then contain albumen, or for a long time contain none at all. The description of the symptoms of simple contracted kidney disease will give me the opportunity of showing the entire dependence of the albuminuria, in this form of disease, upon the blood pressure, and of illustrating it by some striking observations. It appears to me even worth while to narrate one such case now.

CASE IV.—A. L., a journeyman wheelwright, twenty-one years of age, was admitted into my Clinic on June 5, 1872. He complained of fugitive pains over his chest and back, which had lasted already for six months. Now and then he had had some dyspeptic difficulties, and those about him had complained of his breath having a disagreeable odor.

The physical examination of this strong, well-nourished young fellow was as follows: Heart's apex-beat in the fifth intercostal space in the mammary line: action strong, and more widely diffused than is ordinary; præcordial dullness enlarged upwards and to the left; heart-sounds loud and clear, the second sound over the aorta especially accentuated. Radial pulse tense and hurried; the urine he passed directly after his admission contained albumen; that collected the following morning—the night urine—had none in it. During the four months he remained under observation, this varying condition of the urine, according as the patient was up or in bed, was maintained. If he was allowed to stay in bed all day, the diurnal urine too was free from albumen. Indeed, it chanced, one fine day, that we were able to convict him of having been out of bed, when ordered to remain in it—a fact he was unable to deny—solely through the sudden appearance of albumen in his urine.

Casts, although constantly looked for, were only rarely found in the urine, and were then always of small size and perfect samples of hyaline casts. As to the functioning of the kidneys and their secretory activity (the quantity of urine secreted), neither keeping him quiet nor allowing him to be about, exerted any remarkable difference. The patient excreted invariably large quantities of urine—upon an average of eleven estimates, 1,600 c. ctm. daily (min. 1,000, max. 2,500), while the sp. gravity of the eleven days ranged between 1010 and 1022. He was frequently obliged to get up at night to pass urine; the urine was always clear, of pale yellow color, and feebly acid reaction. Its content of albumen was always small. From the beginning of October his urine became free from albumen by day, although he was up and out in the open air. He was then allowed, at his own wish, to go home, and has never presented himself since. His weight increased considerably while he was under observation; the cardiac symptoms remaining unaltered.

Albuminuria, associated with symptoms of increased arterial tension, and hypertrophy of the left ventricle, for which no other explanation can be found, may be accepted as evidence enough to establish the diagnosis, which I did in this case, of contracted kidneys. The invariable fact that the patient, while out of bed and going about, passed albumen, and while quiet in bed passed none, admits, it appears to me, only of the explanation that the whole arterial blood pressure, and consequently that upon the renal branches, is increased above the normal by exercise. And in entire harmony with this was the pulse frequency, which, when he remained in bed, was always less than when he was about, being then from 72 to 76, whereas when he was upright, it numbered from 90 to 96, and even sometimes 100 beats per minute.

The urinary secretion in most cases of amyloid disease of the kidneys, conducts itself just in the same manner as in simple contraction—that is to say, it is watery, of low sp. gravity, and contains constantly small quantities of albumen. And for just the same reason we are prompted to expect that the overflow of albumen into the urine is due to abnormal increase of blood pressure upon the glomeruli. The pathological examination of amyloid kidneys teaches us that this degeneration always takes place first, and most especially, in the capillaries of the glomeruli, whose walls become thickened, reaching in many of them to the extent of actual obliteration of their channels, so that during life they must be as impermeable to the blood stream as they are after death to the entry of an injection fluid. But the parts of the filtering apparatus, which remain pervious, and those arteriolæ rectæ, which spring direct from the main branches of the renal artery, must, as in the simple contracted kidneys, receive the whole mass of the blood stream, which flows through the trunk of the renal artery.

The altered distribution of blood resulting under these circumstances becomes apparent even to the unaided eye when one inspects the mere rough anatomy of the organ. For in the degree in which the large mass of vessels distributed to the cortical substance becomes impervicus to the blood stream, this part of the kidney appears pale and anæmic upon section, and contrasts strongly with the deep red aspect of the medullary portions which remain congested. The filtration of urinary water in the amyloid degeneration continues to take place through the remaining Malpighian tufts, which still retain their functioning powers, and this under almost exactly the same conditions as obtain in the simple contracted kidney—*i. e.*, under abnormally increased blood pressure. The consequence of this is albuminuria.

But whereas in the large proportion of cases of amyloid degeneration the daily measure of urine excreted does not exceed what is normal, while it does so, as a rule, in the contracted kidney, this difference depends plainly upon the circumstance that in the amyloid renal disease there is no general blood tension throughout the whole arterial system, since in this degeneration that hypertrophy of the left ventricle which obtains usually in the other form of kidney disease is not present. But to this we must revert hereafter in the special description of particular forms of renal disease.

It is not to be denied, however, that we meet with cases of pure, uncomplicated amyloid degeneration of the kidneys, in which the urine exhibits totally different characters from those which have been above described; the quantity of secretion, for instance, being abnormally small, its specific gravity high, and its albuminous content enormously large. Whether, in such cases, the difference depends upon an altered permeability of the walls of the vessels produced by the degeneration, I am unable at present to determine. But there are many arguments in favor of this explanation.

We advance now to a fresh question, and one which is of the greatest importance to the inquirer into the causes of albuminuria, namely, *whether the special perviousness to albuminous substances exhibited by the walls of the capillaries within the glomeruli can be altered by any pathological changes that may take place in these walls themselves.*

I do not hesitate to answer this question affirmatively; and towards the vindication of my opinion, I shall first direct attention to the albuminuria so often observed after severe attacks of cholera. This form admits of being classed alongside with that produced in dogs, experimentally, by compression of the renal arteries. (Vide Max Herrmann, l. c. S. 12.) In both cases there is a more or less complete interruption of the arterial blood stream, since, as is well known, in the (pulseless) asphyxia stage of cholera even the larger arteries, which have been divided with the object of letting blood, have been found entirely empty.

Max Herrmann thus explains the albuminuria which ensues upon artificial interruption of the blood stream through the renal arteries in animals: He assumes that the cessation of the arterial blood stream causes a stagnation of the outflow, and that the blood corpuscles will therefore lie stagnant in the capillaries; consequently a great obstruction is offered by these stagnated cells to the blood current directly the compression is taken off the artery and the blood is permitted to circulate through the kidney again. We should be strongly disposed to accept this theory of Herrmann's, in explanation of the post cholera albuminuria, knowing as we do that the blood is excessively inspissated in choleraic disease, and that the conditions that prevail are particularly favorable for the agglutination of the blood corpuscles, if only the capillary thrombosis, which he assumes, had been actually observed. Unfortunately, however, for this doctrine, the examinations made of the cholera kidney exhibit nothing of the kind whatever.

Another explanation is put into our heads by the beautiful experiments of Cohnheim,[1] and his researches into the consequences of a complete interruption of the circulation through various organs in the mammalia. Cohnheim found that when the blood current was interrupted for some little time, the circulation did not become established again when the interruption was removed, apparently because the vitality of the walls of the vessels had been annulled. He further showed that after only a short interruption of the circulation, the organ which had been thus deprived of its nutrition began to swell from the escape of blood plasma into its substance, and then, when the blood stream was readmitted, an emigration of the blood-cells took place through the walls of the vessels. When Cohnheim tightly ligatured upon a piece of leather the artery and vein of a kidney, and, after allowing from one and a half to, at most, two hours to elapse, cut the ligature, the previously pale-gray violet collapsed-looking organ swelled up in a few hours enormously, while its tissues became infiltrated with blood. The kidney then became more than double the size of that on the opposite side, and appeared dark-red colored on its surface and throughout its substance. Looked at under the microscope, all the capillaries and other vessels of both cortical and medullary portions were seen abundantly filled with blood, while a moderate amount of blood-cells were found in the interstitial tissues and the interior of the renal tubes, especially the straight ones. The urine, too, was bloody.[2]

Now my idea of the case is, that in the stage of reaction after severe attacks of cholera, the blood-vessels of the kidney will be in almost exactly the same condition as those of the animals above mentioned, in whom the renal arterial blood stream was experimentally stopped for a short time. Then when the circulation, after its disturbance by the cholera attacks, becomes re-established, blood serum transudes, and the blood corpuscles emigrate through the capillary walls, just as they did in Cohnheim's hands, and albumen appears in the urine, as Herrmann showed by his experiments.

---

[1] *Cohnheim, Jul., Dr.*, Untersuchungen über die embolische Processe. Berlin, 1872, S. 43, ff.

[2] *Cohnheim*, l. c. S. 47.

Moreover, the albuminuria of cholera subsides within a few days, just as Herrmann found it to do in his researches, and similarly the organs (ears of rabbits) upon which Cohnheim experimented shortly recovered their normal condition if only the interruption of the circulation had not been protracted over too long a period. But just as Cohnheim showed that an unduly prolonged interruption of its circulation led to the formation of blood infarctions in an organ, or entirely precluded any re-establishment of the blood stream through it, so, too, we find, after the more severe attacks of cholera, that only scanty quantities of bloody urine are for some time excreted; or else, with the establishment of the reactionary stage, no urine at all is secreted, and in such cases, after death, extensive bloody infarctions are discovered in the kidneys.[1] It might be objected to this view of the etiology of the albuminuria we meet with in cholera, that nothing analogous to what we have described is discovered in any of the other organs of the cholera patient, although the cause accused of producing this disturbance of function in the kidneys—namely, an interrupted circulation—occurs throughout the body. True; but then let us remember that the circulatory arrangement presents so peculiar an obstacle to the blood stream in no other organ of the body except in the kidney, and that, therefore, a complete arrest becomes more easy within the vessels of the Malpighian tufts than in the capillaries elsewhere.

Now, Cohnheim's experiments show that the walls of the blood-vessels are rendered more pervious to the blood by a protracted interruption of the circulation; but another series of inquiries by the same experimentist teach us, by the best possible proofs, that the perviousness of the capillary walls can be promoted by other causes. I refer to Cohnheim's experiments upon inflammation. Cohnheim himself, at the conclusion of his first work upon inflammation and the formation of pus,[1] had already intimated that the secreting vessels, the glomeruli, must conduct themselves, in inflammations of the kidney, just as do the blood-vessels in other organs which admit of being examined under the microscope while they are inflamed, as, for instance, the mesenteries of frogs and young mammalia. In fact, both the pathological appearances of inflamed kidneys and the characters of the urine secreted by them speak positively in favor of this, that just as blood plasma and white blood-cells are effused in the inflammation excited by cantharides on the skin, so, too, an inflammatory exudation takes place through the walls of the vessels in the glomeruli in inflammation of the kidneys, and becomes mingled with the urine secreted by them.

---

[1] *B. Reinhardt* und *R. Leubuscher*, Beobachtungen über die epidemische Cholera. Virchow's Archiv. Bd. 2, S. 500.

Whether an inflammatory alteration in the kidney tissues runs an acute or chronic course, we recognize it after death by swelling and finely granular clouding of the epithelium in the urine tubes, combined with more or less abundant accumulation of white blood-cells in the interstitial connective-tissue spaces. But besides this, there is, as many authors have observed, an unwontedly distinct protrusion of the glomeruli produced by their repletion with blood. Colberg—who, from his examinations of chronically inflamed kidneys, had had his attention especially directed to the size of the Malpighian bodies—showed me some preparations, in which the glomeruli were several times larger than those of sound kidneys. Virchow[2] has said that the coils of the glomeruli in chronic nephritis often appear thicker and more cloudy than they ought to, and that, when the section is treated with acetic acid and rendered transparent, three or four times the normal number of nuclei become visible, and the thickened wall of the capillaries stands out prominently.

It has been customary to regard the enormous distention of the capillary coils of the glomeruli in inflamed kidneys solely as the result of congestion of the efferent vessels, in consequence of the compression of these by the swollen parenchyma. But, in my opinion, there is no reason why we should not consider this state of the capillary vessels in the Malpighian tufts as inflammatory, just like what obtains in inflammation in capillaries elsewhere. Indeed we are all the more inclined to this idea, since we do not find this condition of the Malpighian capillary coils produced by other causes that obstruct the blood-flow from out of them and lead to albuminuria—as, for example, in the cyanotic induration of the kidneys. The walls of the capillaries, when stimulated by the influence of inflammation—arise this from what cause it may—become more pervious to the transudation of substances than they are in their normal state. Wherefore I believe the opinion is perfectly correct that it is not an altered blood pressure only, but also an alteration in the actual filtering apparatus of the glomeruli, which is and must be the source of the albuminuria in the inflammatory affections of the kidneys.

---

[1] Virchow's Archiv. Bd. 40, S. 77.

[2] Gesammelte Abhandlungen, S. 485. Anmerkung.

This view obtains entire confirmation from the nature of the secretion furnished when the renal vessels are thus abnormally affected, since the character of the urine serves to distinguish it in a marked manner from that secreted by kidneys which are merely in a condition of congestive hyperæmia. Thus in the urine furnished by inflamed kidneys we discover albumen and fibrine, the elements of the blood-plasma, and find white blood-cells, as a rule, in the sediment—the latter appearing sometimes in huge numbers, and being mixed with large quantities of red blood-cells in the acute cases of nephritis of decided severity. But the elements above mentioned are those very constituents of the blood which are in the habit of passing through the walls of the blood-vessels under the stimulus of inflammation in any organ, and which, according to the locality in which they transude, collect either in the serous sacs as free exudations, or accumulate in the interstitial tissues, producing swelling of the parts in which they occur. Now in inflammation of the kidneys it is plain that both these events ensue; a part of the free fluid exudation escapes into the urine through the renal tubes, the rest—that portion which we may surmise is furnished by the intertubular capillaries—passes into the intertubular tissues and into the epithelium of the renal tubes, producing swelling in both situations.

The altered aspect of the epithelium thus induced has been regarded by many writers as the explanation of the albuminuria in inflammation of the kidneys. Substantial reasons, however, for this view are not communicated, and still less is any attempt made to combat the arguments which so remarkably favor the idea that the albumen in acute nephritis is derived from the capillary coils of the glomeruli. One can always suppose that the epithelia of the renal tubes furnish a part of the albumen—that part which, under the stimulus of inflammation, is excessive and beyond the needs of their nutrition; the share which they take up, but cannot appropriate, and so shed out again into the secreted urine, adding just thus much to its albuminous content. But then the fact has never yet been proved, and indeed obtains no confirmation, nay, little likelihood, from what we know of the epithelia of other parts, under analogous conditions. It would enter no one's mind to regard the albumen contained in a pleuritic exudation as a kind of excretion furnished by the epithelia of the pleura, or as an albuminoid metamorphosis of these.

Compared with all other causes of albuminuria, inflammation of the kidneys, and especially that form which runs a chronic course, furnishes the largest percentage amount of albumen to the urine. In such cases the quantity of albumen can reach five per cent., which is more than half the amount contained in normal blood serum. The inflammatory exudations poured into the serous cavities of the body are, of course, considerably richer than this in albumen; but then there is no watery secretion, no urine, mixed with them.

c. Hæmaturia.

The formed constituents of the blood, and prominently the red blood-cells, pass into the urine, and may be accepted as a symptom of kidney disease, although they certainly occur more rarely as a symptom than does the escape of serum albumen alone.

So far as regards the white blood-cells, we have already noticed in the previous section that these can migrate through the walls of the capillaries of the Malpighian tufts, together with the urinary water; but they may also become mixed with urine which has been secreted from the kidneys, upon the transit of this through the conducting channels, in consequence of inflammation of the mucous membrane, or through the bursting of some abscess close at hand.

I have further remarked that any urine containing white blood-cells must also be albuminous, because when the white blood-cells thus migrate, albumen, too, will transude through the walls of the blood-vessels; now exactly the same thing may be vouchsafed of hæmaturia, when red blood-cells are mixed with the urine. The red corpuscles, too, may escape from the vessels of the kidneys into the urine, or they may become mixed with urine that was secreted in a normal state, but into which blood afterwards escaped, either from the calices, the ureters, the bladder, or the urethra.

But when the red cells pass into the urine, a notable quantity of albumen accompanies them. Bloody urine is invariably albuminous. At the same time one must consider the fact, established by Cohnheim in his experiments upon the process of inflammation and that of mere venous stasis, that in inflammation, besides the white cells, a far larger quantity of albumen passes into the urine than is pressed out, together with red blood-cells, through the capillary walls, under conditions of venous stasis. Hence it does not follow that a urine which contains a pretty large quantity of red blood-cells should be very highly albuminous; indeed, my own experience tells me that the urine of hæmaturia, as a rule, contains only a small quantity of albumen—indeed, a far smaller quantity than one finds in the pale, bloodless secretion furnished in chronic renal inflammation. The fibrinous constituents of the blood plasma, after they have escaped from the walls of the vessels, into the urine, in hæmaturia, coagulate into firm clots, which entangle the red cells or other formed elements with which they may lie in contact at the moment of coagulation. The form and quantity of these coagula excreted with bloody urine vary with the seat of the bleeding and the amount of the blood. Thus, the more copious the bleeding, the more numerous they will be, and, if the bleeding take place in a roomy part of the urinary apparatus, the larger will be the size of the clots. In respect to the size and shape of the coagula, these, besides being in some measure determined by the density or thinness of the escaped blood, will be governed by the form of the cavity in which they are moulded. Thus in the bladder huge blood-clots may be formed, which cannot pass out through the urethra without suffering diminution of size, either by compression as induced by contraction of the bladder, or by mechanical breaking up with instruments passed up into the bladder.

Firm, rounded clots, corresponding exactly in length and diameter with that of the distended ureter in which they were formed, occur when merely pure blood is poured out into the ureters, an event possible enough when the entire pelvis of a kidney is blocked up with coagula or with masses of cancer; and the passage of these ureter-shaped thrombi, measuring often more than a finger's length, and being of about the size and shape of a lumbricoid worm, has given rise to errors of diagnosis, to the assumption, for instance, of the presence of entozoa (strongylus, for example), whose very existence at all in man is disputed.

In hemorrhages from the pelvis of the kidney I have often seen blood-clots in the urine which exactly took the shape and size of the calices.

If the urine be mixed with blood derived from the substance of the kidney, the quantity of this is, as a rule, so small that the clotting of the fibrine takes place within the renal tubes and before the renal pelves are reached. The coagula then give evidence, by their form and shape, of their point of origin; they make casts of the channels whence they were derived, and, although themselves so small as to escape a naked-eye examination, they may be discovered in great numbers in the urine sediment when put under the microscope. It very often happens, however, in renal hæmaturia, that, although the urine has a characteristic blood color, the actual quantity of blood contained in it is so insignificant that but scanty coagula are formed. Still, as a fact, which I must strongly insist upon, I may remark that the mere quantity of red blood-cells mixed with the urine should be accepted as no measure whatever of the quantity of fibrine that has transuded the capillary walls in their company. The actual cause of the hemorrhage is far more important than anything else in explaining the quantity of fibrinous clots which a bloody urine contains. Thus I am enabled to state positively that the coagula present will be few, if the hæmaturia is produced by venous stasis; whereas, if the bleeding be an accompaniment of inflammation of the kidneys, a large number of renal casts, entangling blood-cells, will be excreted.

Such profuse kidney bleeding as leads to the formation of large clots in the pelvis of these organs, or in the bladder, I have only seen in the rare instances of traumatic lesion of one kidney (for example, crushing or rupture of the right kidney, the result of a kick), and in cases where a highly vascular cancer has grown into the pelvis of the kidney.

We are usually able to recognize the presence of blood in urine immediately by its characteristic color, a color necessarily imparted to this fluid; but, as we have previously remarked, this color will vary considerably, from that of pale, raw meat up to brown-black, in accordance with the quantity present. Bloody urine is more often sooty or dark-colored than bright red, and the liquid is more usually cloudy than clear. Very small quantities of blood do not alter the color of urine much; but even here one can often recognize the presence of blood by the look of the sediment, if only the urine be allowed to stand in a funnel-shaped glass, terminating in a point at its bottom, that the blood-corpuscles may subside into it (Neubauer). The surest and most trustworthy way, however, of recognizing blood in urine, is by employing the microscope, and distinguishing the cells in the sediment.

We sometimes meet with cases in which, although the urine looks deeply blood-stained, we find no red cells at all to explain the color and only a few uncolored corpuscles under the microscope. Now this depends upon one of two things: either the blood-pigment has escaped, the red cells having suffered destruction while still within the capillaries, and the coloring matter having escaped together with the other constituents of the urine; or the red cells primarily excreted with the urine have subsequently broken up in this, and have yielded their coloring matter to it either in an unchanged form, or after it has suffered conversion into hæmatin. In urines thus blood-stained, and which have been subjected to examination without further treatment, I have often observed that peculiarity of dichroism of which I have already spoken.

The surest test that we possess for blood-coloring matters dissolved in urine is that by means of the spectroscope.[1] Heller's test, too, can be employed to discover blood or blood-pigment in urine. It consists in the reaction furnished when caustic soda solution is added to urine, and boiled with it in a test-tube. The earthy phosphates precipitate and entangle as they settle the hæmatin (resulting from the decomposition of the hæmoglobulin), which is thus carried in the sediment to the bottom of the tube, and presents sometimes a brick dust and at other times a bright red color, the fluid often exhibiting a dichroistic green color to reflected light.

Whenever blood appears in the urine, it is important to decide from what part of the urinary apparatus it has been derived, whether from the kidneys or from the mucous lining of the passages; and here, apart from all the other circumstances which have to be taken into account, we find that the size and shape of the blood coagula especially assist us in arriving at a diagnosis, affording as they do precise symptoms for determining the existence of disease either in the pelvis of the kidney or in the bladder. It can be safely pronounced that the large, readily distinguishable clots and the blood which led to their formation could only in the very rarest instances (injuries) be derived from the kidneys and the renal vessels proper. In true hemorrhage of the kidney the coagula are formed inside the renal tubes, and present themselves as cylindrical casts, betokening their place of origin; they become apparent only beneath the microscope, and are then seen to entangle blood-cells in their interiors and to be accompanied by other free blood corpuscles.

When there are no blood-cells, but the coloring matter of the blood only is present in the urine, this will have been derived from the vessels of the kidney, having been excreted from them together with the rest of the constituents of the urine. And then it can happen that the hæmoglobin may in part spontaneously decompose, and hæmatin appear alongside the remaining unaltered portion. In some deeply blood-stained urine passed by a typhoid fever patient and containing, so far as could be ascertained by a microscopic examination, no blood corpuscles, Immermann[1] proved, by aid of the spectroscope, the presence of hæmatin as well as hæmoglobin.

---

[1] Vide *Neubauer*, l. c. S. 123–126.

As causes of renal hemorrhage, apart from the effects of injuries and cancerous growths, we may reckon all those affections of the kidney which also entail albuminuria, namely, overrepletion of the renal vessels, and inflammations of the kidney substance. Simple overfulness of the vessels may arise in the form of active congestion, like that produced by such irritant substances as oil of turpentine or cantharides. N. Sowloff,[2] of Botkin's Clinic, communicates a marvellous case of repeated hemorrhages from the kidneys of short duration, depending upon the action of cold upon the integuments of the body, a case which admits of no other explanation but that of collateral blood fluxion. The mere venous congestion, or, as it is called, passive hyperæmia, can reach such a grade that the blood is squeezed per diapedesin through the walls of the capillaries into the renal tubules. This is the case, for example, when the obstruction to the outflow of the blood is situated either in the renal vein itself or in the vena cava ascendens, as when a thrombus forms in either of these vessels, while the arterial blood supply to the kidney remains uninterrupted. The venous congestion that follows upon cardiac insufficiency but rarely leads to a distinct mixture of blood and urine; but, on the other hand, hæmaturia occurs in heart disease, and by no means rarely, as a symptom appointing hemorrhagic infarction of the kidney, the result of embolism; but the hæmaturia then is always transitory and short in its duration.

Copious and persistent renal hemorrhages are doubtless commonest in acute diffuse nephritis, and then the blood indisputably escapes into the renal tubules from the capillaries of the glomeruli, altered as these are by inflammation.

---

[1] Ein Fall von Hämatogenem Ikterus. Deutsches Archiv für klin. Med. Bd. 12, S. 502.

[2] Berl. klin. Wochenschrift, 1874, No. 20.

The hæmaturia which occurs in so-called states of dissolution of the blood—for example, in scorbutus, in the morbus maculosus of Werlhof, and in hemorrhagic small-pox—is certainly only in quite exceptional cases to be regarded as a true renal hemorrhage. In most of the examples of Werlhof's purpura disease, and in the many cases of hemorrhagic small-pox in which I have examined the urine, the source of the bleeding was generally found to be either one or the other kidney pelvis, or the blood was derived more rarely from both, and from the bladder as well. The passage of hæmoglobin into the urine without blood-cells ensues, as above noticed, from dissolution of the red cells, which takes place even before they leave the renal vessels. As causes of this dissolution or destruction of the blood corpuscles, the graver forms of fever, and the action of particular poisons may be mentioned. Thus it has been observed in abdominal typhus, septicæmia, and poisoning by phosphorus, by arsenic and its compounds, and by sulphuric acid.

5. *The Formed Elements of the Urine in Renal Disease.*

Of scarcely less importance for purposes of diagnosis than the chemical analysis of the urine in renal disease comes the microscopical investigation of sedimentary deposits, since we can thus obtain acquaintance with the formed materials excreted in the urine.

Under this category of formed elements we encounter a vast variety of substances: first, those which are at all times contained in normal urine, but which from their excessive quantity or abnormal form are calculated to induce disease of the urinary apparatus—as, for instance, uric acid or oxalate of lime, appearing in crystalline shape while still within the urinary channels, or the crystalline or amorphous deposits of earthy phosphates, or the rarer and to healthy urine alien crystals of cystine, which, like the rest which we have mentioned, may lay the foundation of concretionary formations. Secondly, we meet with elements whose mere appearance in the urine can only be accepted as certain evidence of abnormality, appointing either disturbance of the kidneys' secretory faculties, or indicating actual disease of the urinary passages; such are blood-cells, and epithelium derived from various parts of both the secreting and the conducting apparatus. And, lastly, we sometimes find what are known as casts and recognized as pathological formations derived from the renal tubules.

In urine which is just passed and which is perfectly fresh, and not as yet altered by exposure to air, the presence of crystals of uric acid, or of oxalate of lime, or of cystine, means nothing so far as the kidneys are concerned, but signifies that these substances, secreted by the kidneys in soluble form, have been converted into solid form within the urinary passages, and in this form they can give rise to concretions, and so lead to inflammation of the passages, and, secondarily, of the kidneys themselves.

Each one of these substances in crystalline form may be discovered in urine which has emanated from perfectly sound kidneys; but they may all of them be found also in the urine derived from diseased kidneys.

But if a urine directly after it is passed is cloudy, from a separation of its earthy phosphates as a fine precipitate, and if such a urine when perfectly fresh also contains crystals of ammoniaco-magnesian phosphate, it betrays invariably ammoniacal decomposition within the urinary passages, either as a result of inflammation in the mucous membranes lining these, or in consequence of the penetration of some fermenting substance into the bladder.

The urine of a man in health may be alkaline, as happens after the excessive ingestion of some alkaline salt, but the earthy salts will then still be held in solution and the fluid continue for a while transparent, since the urinary secretion normally, and, as a rule, contains enough carbonic acid to hold its earthy salts in solution; but if this carbonic acid be dissipated by boiling, the earthy salts fall at once as a white precipitate. But under normal conditions, the urine as it is excreted should never contain carbonate of ammonia.

The demonstration of the above-named crystals in any urinary sediment becomes therefore only of practical importance when the question asked is: first, is there a suspicion of the existence of some concretion in the urinary tract—in the pelvis of the kidney, or in the bladder; and, secondly, if such concretion exist, of what substance is it likely to be composed? As to the condition of the kidney, we can conclude nothing from the presence or absence in the sediment of the urine of the materials out of which a calculus might be formed.

We must reserve for fuller discussion in the section of this work, in which it is intended to treat of the mode of origin of urinary concretions, the description of the form and other characters of these sediments.

In a previous portion of this treatise we spoke of the special value that can attach to the presence of red and white blood corpuscles in the sediment of urine, and showed how the information thence derived could be applied to the diagnosis of disease in the kidneys and urinary passages; still we may insist here that such a quantity of pus as can form a compact sediment, such, too, as we meet with in inflammation either of the kidney pelvis or of the bladder, never happens as the result of an affection of the kidney only, if we except those rare examples where a renal abscess bursts into the pelvis of the kidney.

In the severest and most diffuse inflammations of the substance of the kidney, the pus cells furnished form but a small share of the sediment of the urine, which owes its principal opacity to other precipitating matters. The opacity of the urine in such cases, besides what is due to amorphous deposits of urates and earthy phosphates, is derived from epithelium, which may come from the lining membrane of the renal pelves or from the ureters, from the bladder, the urethra, or the vagina. The presence of epithelium in the urine in any notable quantity implies invariably a pathological shedding or moulding from some matrix tissue. If the individual cells exhibit no departure from their normal condition, the desquamation may be the sequence of some entirely temporary cause, as, for instance, simple hyperæmia. But, on the other hand, if the epithelium is rendered opaque by the presence of fatty particles, or appears in fragmentary pieces, and any large quantity of it is found in the urine, it may be assumed that the parts from which it was derived are the seat of some degenerative process, of inflammatory or other origin. As to the nature, however, of the degeneration, the other characters of the urine must furnish the requisite information. But we come now to the tubal or cylindrical casts, which are of greater importance towards the diagnosis of renal disease than any of the sedimentary matters which have been hitherto noticed.

Urinary Casts or Cylinders.[1]

The cylindrical objects, derived from the renal tubes, and which, according to my experience, are never formed or shed in a perfectly normal state of health, certainly ought not to be accepted as certain evidence of structural alterations in the kidneys; on the other hand, they are by no means always or invariably observed in kidney disease. It remains a disputed matter who was the first person who saw and described these objects. In a prize essay, which obtained the reward for which it contended, written and recently published by Dr. A. Burkart, entitled "The Urinary Cylinders, with Special Consideration of their Value in Diagnosis," Berlin, 1874, I find a quotation from the Correspondenzblatt rheinischer und westphalischer Aerzte, Jahrgang 1843, from which it appears that Prof. Nasse, of Marburg, did not merely notice cylindrical casts, but referred their formation to the renal tubules. Vigla and Rayer had previously given good reason for believing that they had met with these same objects in their microscopic examinations of the sediment furnished by albuminous urine. But Henle[2] was undoubtedly the first person who after, in the year 1842, he had found casts in the urine of a person afflicted with dropsy and albuminuria, discovered the same objects in the tubules of the diseased kidney derived from the same individual.

---

[1] I have adopted the term cylinders, which is made use of by many writers, without having ever been able to convince myself that these objects, with the exception of the true epithelial tubular casts, really are hollow; but I cannot bring myself to designate the other forms of cylindrical casts of the renal tubes as tubular casts.

[2] *Henle*, Zeitschrift fur rationelle Medicin. Bd. I. S. 68.

From this date the attention of medical men has remained directed to these objects. They are found *in situ* in the urinary tubules in the pathological scrutiny of diseased kidneys, often in considerable number, not only in the larger straight tubes, but also in the smaller sling-shaped down-loopers and in the curling tubes of the cortical substance. In correspondence with this, one can distinguish both broad and narrow casts in all urine sediments. They measure from 0.01 to 0.05 of a millimetre in diameter, and the length of both sorts may vary notably. At times only short fragments are discovered, being scarcely any longer than they are broad; at others one finds cylindrical casts a millimetre or more in length. Thanks to the kindness of my colleague, Heller, I possess a large number of microscopical measurements of casts, the results of which are given in the numbers above stated.

It is not, however, only in their size that these casts differ so much one from another; they present other characteristic peculiarities which compel us to distinguish various forms of renal casts.

Here, in the first place, I must distinctly specify two kinds of cylindrical formations as to whose nature and significance no difference of opinion at all exists. In the first category I place the so-called *epithelial casts*, which consist of simple pipes formed of the epithelia of renal tubes stuck together. In the course of acute inflammation of the kidneys, the epithelial linings of the renal tubules may be shed in their natural continuity, to appear as cylindrical casts in the urine. The source and meaning of this kind of casts, which, as it happens, are not often seen, speak for themselves. Nor is the interpretation set upon those I place in my second category less clear. These are the *blood-casts* (Fig. 1) previously noticed by me, which also have a cylindrical form. They are derived from the renal tubules in hæmaturia, and are then excreted in the urine. They consist of coagulated fibrine, and entangle for the most part such a quantity of red corpuscles in them as to appear under the microscope perfectly dark and opaque, although the separate blood-cells may still distinctly be made out.

In contradistinction to these two kinds of cylindriform casts, I must distinguish certain other forms that arise from the renal tubules and are shaped after them, but which are far more commonly met with and accompany quite a variety of pathological conditions. These deserve the appellation of urinary cylinders in the narrower sense of the term. I believe that I am right in thus describing them, and do this advisedly, so as not to prejudge the still doubtful nature of the objects themselves. These casts, too, exhibit such characteristic differences that we are compelled to describe separate varieties of them.

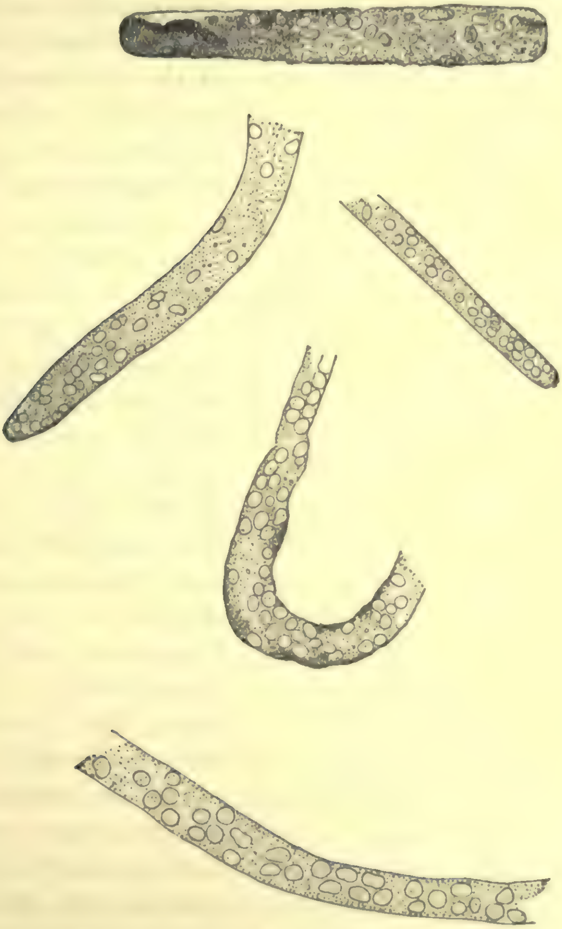


FIG. 1.
Blood casts from the urine of a case of acute parenchymatous nephritis.

First, then, we meet with perfectly homogeneous casts, transparent, and so colorless that their outlines are only with difficulty rendered apparent in the fluid surrounding them—*hyaline casts.* Their discovery is facilitated by adding solution of iodine in iodide of potassium, or a not very concentrated carmine solution to the microscopical preparation, by which means they are stained yellow or red respectively. They are for the most part narrow; their ordinary glass-like aspect is occasionally varied by a light streaky shading; some of them are rendered more or less distinct by a finely granular opacity or by the presence of very small oil drops. Then, frequently these narrow hyaline casts are not of quite the same diameter throughout, but tail off at one extremity. Nearly as often they present some bend or bowing of themselves, or a slight indentation at some one part. Less usually I have remarked that some of the broader specimens exhibit a bifurcation at one end, and others one or more side indentations; this, however, is an idiosyncrasy only of the broad examples.

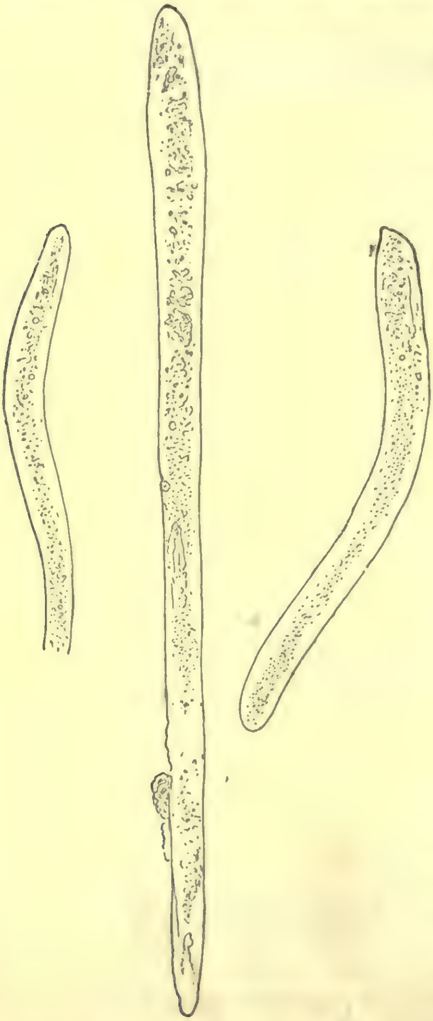


FIG. 2.
Hyaline casts from the urine in contracted kidney.

Fig. 2 delineates both the broad and narrow varieties.

The second variety of urinary casts we have to describe appears to consist entirely of granular masses, and they are, therefore, less transparent to light and appear under the microscope much darker than those we mentioned first. Most of them are rather broad, and many of these *dark granular casts* are indented at their sides, and this, too, sometimes at pretty regular intervals, as if they were composed of several pieces stuck together, or were on the eve of breaking up into such pieces. At their extremities and here and there at their sides they often look worn or eaten out, as if they were in the act of crumbling to pieces.

Fig. 3 gives representations of them.

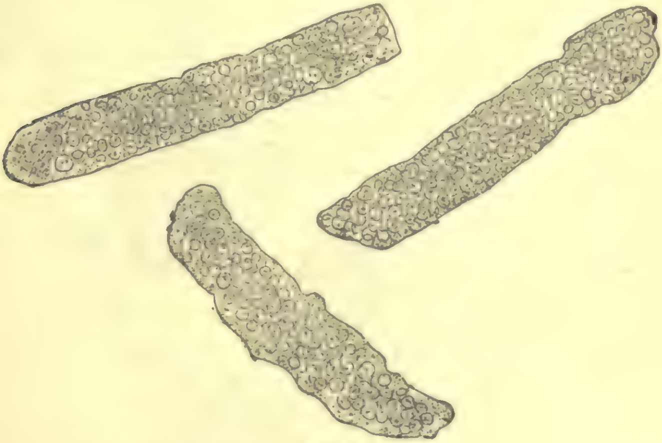


FIG. 3.
Granular casts from the urine in secondary contraction of the kidneys.

The third variety of casts consists of certain forms which always exhibit under the microscope a peculiar glistening aspect, and often have a distinct yellow staining — *the waxy casts.* Regarded apart from the matters which are occasionally attached to them, they are quite homogeneous, but are easily distinguishable from the hyaline casts above described, which were also originally homogeneous, by their highly refractive properties and also by their slight coloration. Among them we encounter some specimens which have a considerably greater diameter than have any of the kinds previously described. They are sometimes even broader than the tubules in their ordinary condition at their debouchment in the pyramids.

Fig. 4 gives a representation of these casts, showing their thickness compared with that of the other varieties; for they are all drawn on one scale.

All these forms of casts may have substances attached to them, as epithelial cells or fragments of cells from the tubes, free white or red blood corpuscles, amorphous deposits of urates, or, more rarely, crystals of uric acid or oxalate of lime.

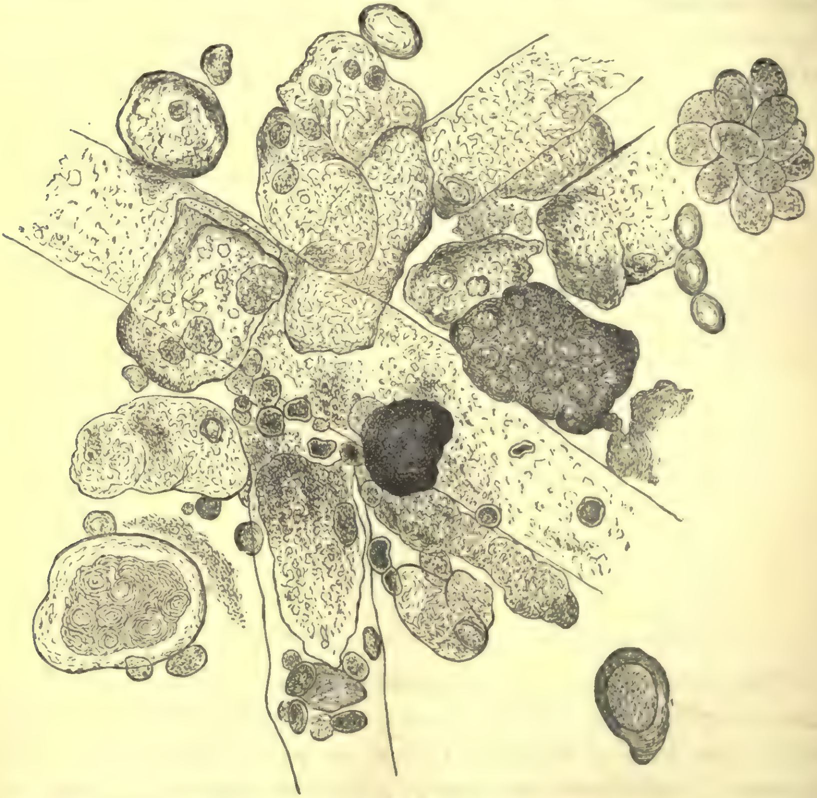


FIG. 4.
Broad, waxy, highly refractive casts from the urine in amyloid degeneration of the kidneys, from a case of phthisis.

Turning now from these forms of casts which emanate indisputably from the tubules of the kidney, we must lastly distinguish some others also occasionally found in the urine, and which possess at least some likeness to those which we have already described. Whereas, however, true cylindrical casts always convey the impression of being solid bodies under the microscope, these have more the look of strips of ribbon. Their edges run parallel to each other; their ends are either frayed out or tattered; or their ends are pointed at one edge, or, finally,

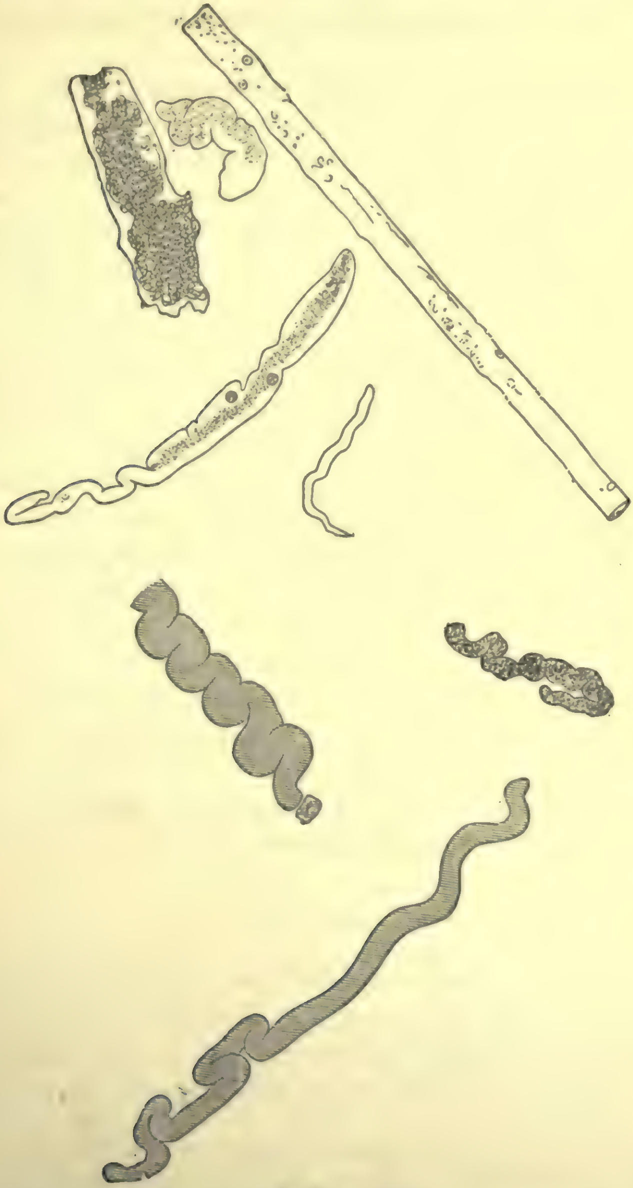


FIG. 5.
Cylindroids and hyaline cylinder casts from the urine of scarlatinal nephritis.

folded or twisted up in a spiral. These flat, ribbon-like objects are often considerably longer than even the longest true renal casts, and are usually as broad, if not broader, than the very thickest; they are homogeneous, colorless, and very pale, and I never saw any renal epithelium or crystals of any kind attached to them.

Fig. 5 gives drawings of these cylindriform casts.

Thomas,[1] in his treatise on the microscopical appearances of the urinary sediment derived from scarlet-fever cases, manifestly had these objects before him. The same appearances are seen, however, in other forms of albuminuria. As to the place in which they are formed, we know at present nothing certain. Thomas considers it as an unquestionable fact that most of these bodies which he calls *cylindroids* have their origin in the renal tubules. He distinguishes several varieties, and has found them too in urine which contained no albumen. They are so totally different from the cylindrical casts previously described by us, that they must admit of some completely diverse explanation. At the same time Thomas thought he had made out some forms of these casts which, in his opinion, should be looked upon as transitional or intermediate between them and true casts.

Lastly, Nothnagel[2] has described a peculiar kind of hyaline cast which he discovered in the non-albuminous urine of icteric patients. These I have often sought for, and once seen in some intensely icteric urine which was free from albumen. They were uncommonly pale, longer, and considerably narrower than any renal casts I have ever seen in albuminuria, and no formed substances, cell fragments, or crystals, stuck to these slender pale threads.

Let us ask ourselves now *what these urinary casts consist of, how they originate, and what it is that their presence betokens.*

Henle, when he first discovered them in the urine, and after he had traced them back to the kidneys, took them for fibrine, and considered that they were derived from the blood plasma excreted with the proper urinary secretion into the tubules, and that they became coagulated and moulded within these. This, too, has been the opinion held by most writers on the subject during recent times, and hence the term fibrinous has come into general use to describe every kind of true urinary cast, without any excepion being taken to it. But when the fibrinous nature of these coagula became doubtful, and after several other descriptive terms besides fibrinous had crept into use, the learned Swede, Axel Key, who, as is well known, had distinguished a great many varieties of casts, pointed out how completely every kind of renal cast differed from fibrine in its chemical reactions. Rovida, of Milan,[1] is the most recent author who has thoroughly investigated the chemical nature of the renal casts. He distinguishes only three varieties of them: *the colorless*, *the yellow*, and *the epithelial*, among which last he obviously includes the dark granular form described above by me. His investigations, so far as the colorless and yellow casts are concerned, led to the conclusion that these cannot consist either of fibrine or of any protein substance, nor can they be formed of gelatin, chondrin, mucin, or hyalin. Then, both the forms above named differ chemically from each other: the colorless ones dissolve in pure water, aided by heat; whereas the yellow do not. The latter resist the action of reagents for a much longer time than do the former. Both, it is true, possess certain of the characteristic properties of protein substances, so that Rovida takes it for granted that they are derivatives of albumen, or are, as Gorup-Besanez would call them, albuminoid substances. Fuller information upon the conduct of both kinds of casts with chemical reagents may be found in Rovida's work, which amply justifies the negative conclusions at which he arrives; and there the question remains at the present time.

---

[1] Klinische Studien über die Nierenkrankheiten bei Scharlach. Archiv für Heilkunde. Bd. II. 1870, S. 130.

[2] Harncylinder beim Ikterus. Deutsches Archiv für klin. Med. Bd. 12, S. 326.

The effect of these inquiries into the chemistry of the urinary casts has necessitated an alteration of the views previously entertained upon the mode of their origin. Key and Oedmansson, the two Swedish observers, have been the most decided opponents of the idea that all forms of urinary casts originate in blood fibrine excreted with the urine. While Key admits that certain of the varieties of casts which he describes originate in a degeneration of the renal epithelium, and in a smelting down of these cells into a homogeneous mass, he allows that certain others are secreted by the epithelium. Oedmansson, on the other hand, is of opinion that every cast should be regarded as a product of secretion furnished by the epithelium. Ottomar Bayer,[1] of Leipzic, who tried to discover the process of cast-formations, from the study of anatomical preparations, concluded that each variety of cast was formed by the moulding together of different kinds of degenerated epithelium. Bayer finishes his work with the following conclusion: "In spite of the whole epithelial substance escaping in the form of cylindrical casts, still an epithelial lining of the tubules is commonly preserved for some time, and Key endows this epithelial lining with the liveliest powers of self-regeneration upon the broadest evidence; and, in confirmation of this, I have found, myself, that losses of substance, which threatened to be considerable, were obviated by repairs instituted by such cells as still remained, the tunica propria and intertubular connective tissue in many chronic cases themselves participating in the processes of regeneration."

---

[1] Ueber das Wesen der Harncylinder. Moleschott's Untersuchungen zur Naturlehre des Menschen und der Thiere. Bd. 11, S. 1.

The origin of the colorless hyaline casts, as a secretion from the epithelial cells, was later on defended by Oertel;[2] while Rovida[3] attributed both the colorless and the yellow hyaline casts to a similar source. Both investigators observed the following appearances in anatomical preparations: the lumina of the renal tubules were, in Oertel's cases, filled with hyaline spheroidal bodies, drops of plasma, which were intermingled with a finely granular friable substance, into which the epithelial cells had become transformed, and in which no nuclei were apparent, and seemed to form with it a single mass. Upon further examination he found spots where the degenerated epithelium was completely separated from the walls, and where a solid cast or pipe-like mass was formed out of them—a mass, however, in which some well-preserved spheroidal bodies of plasma could still be made out. Rovida, examining during life the urine of a case of chronic diffuse nephritis, found transparent as well as yellow urinary casts, and several highly refracting yellowish scales, fragments of broken yellow casts. After death, when the kidney was examined, and fine sections of the cortical portion were colored and put under the microscope, the curling tubes were seen to be completely filled with granular opaque epithelium, in which the nuclei were scarcely visible; the interiors of the tubes were filled with small homogeneous, or for the most part finely granular spheroids, which corresponded exactly in color and refractive properties to the yellow casts, and, after coloration with hæmatoxylin or carmine solution, exhibited no trace of nuclei. In many of the tubules one saw these bodies as half spheres or portions of long drops protruding from the bodies of epithelial cells into the lumen of the tube; and in some of the tubules they were so placed and compressed against each other as to present many flat facets and form irregular polygonal shapes, while in other parts their contours as spherical bodies were entirely lost or only just to be made out. Stuck together into a single mass, they formed a cast, which more or less completely filled the calibre of the urinary tube. Thanks to the kindness of my colleague, Heller, I am enabled to give a drawing of a preparation which illustrates Rovida's observation of the mode of origin of these yellow casts. It came from an atrophied kidney with pronounced amyloid degeneration of its blood-vessels.

---

[1] Ueber den Ursprung der sogenannt. Fibrincylinder des Urins. Archiv für Heilkunde. 1868, S. 136.

[2] Experimentelle Untersuchungen über Diphtherie. Deutsches Archiv f. klin. Med. Bd. 8, S. 292.

[3] Ueber den Ursprung der Harncylinder. Moleschott's Untersuch. l. c. Bd. 11. S. 182.

After this it can hardly be disputed that some casts arise as a species of secretion from the epithelium; but of a surety every form of cast is not made in this way. The dark granular casts, indented at somewhat regular intervals, originate, I take it, as A. Key has indicated, directly by an agglomeration of degenerated epithelial cells. At the same time I must insist that in each case in which I have microscopically examined fine sections of the diseased kidneys whose renal tubes were blocked up with casts of this kind, the walls of the tubules which were distended by these abnormal contents invariably exhibited an epithelial lining. Key and Ottomar Bayer explain this fact upon the theory that there is a reproduction of epithelium replacing that which has already been shed.

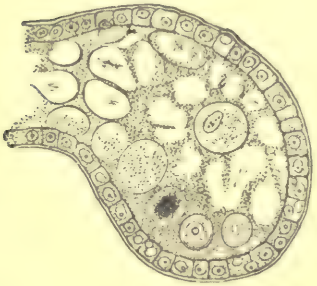


FIG. 6.

Waxy highly refracting drop-like bodies formed in the interior of a urinary tubule, which still retained its lining of unaltered epithelium. Atrophic amyloid kidney. (Heller.)

Clinical experience, however, compels me to assume a third mode of origin for true casts, or rather, for particular forms of casts, to hold fast to the theory of origin which was originally propounded, namely, that they are formed by a coagulation of the albumen or its derivatives excreted with the urine. The casts to which I refer are the homogeneous, transparent, lightly streaked, or faintly shaded varieties, or the forms which are so delicately stippled with the finest granules or minute oil drops—in a word, the casts which are most rightly called hyaline. Furthermore, my experience informs me that their occurrence is entirely dependent upon the albumen mixed with the urine. Indeed, the interdependence of the two things is doubly confirmed: first, because these forms are only found when albumen is present; and, secondly, because in by far the larger number of cases the arrival of the albumen and of the casts is simultaneous. It has happened to me, as of course to other writers who have contended for this mode of origin, to find these forms in non-albuminous urine; but this has only been the case when the albuminuria has but very recently disappeared. Still I believe that I have already proved (see Case III.) that the formation and the excretion of a cast need be by no means contemporaneous events; and I also referred, in the same place, to the further changes which these bodies undergo in consequence of a protracted sojourn in the spot of their original formation.

Henle,[1] it is true, has rarely failed to discover these casts in healthy kidneys belonging both to men and to animals, and states that the tubules in which they are formed, as a rule, lack epithelium. But I am at a loss to understand the disagreement between his observations and the results arrived at by most other inquirers. I have already, however, referred to the frequency with which patients affected with severe febrile disorders pass albuminous urine. Now this urine, almost every time I have examined it, has also contained pale hyaline casts, and yet in the kidneys of these patients, if they died, often no pathological alteration whatever could be found, not even cloudy swelling of the epithelium. Could it have been kidneys like these which Henle examined?

I must therefore insist upon the following rule: *the formation of true casts, or renal cylinders proper, never does take place under perfectly normal conditions, and the appearance of any species of cast—be the cause of its formation what it may—is, as a general rule, associated with the excretion of albumen in the urine.* Now bearing in mind that the different kinds of casts most likely have different modes of origin, it will be evident that my proposition receives no opposition in the fact that the percentage amount of albumen and the quantity of casts contained in the sediment, have a direct relation to each other, although not in all, still certainly in the large proportion of cases. In particular instances a great quantity of albumen implies, without further inquiry, that a great number of casts will be present—as, for example, in diffuse nephritis; whereas the watery urines of the genuine contracting kidney, and of amyloid disease, where but a sparing amount of albumen is present, usually furnish us with but few cylinders. We meet, however, with some urines—*e. g.*, in certain forms of amyloid disease—which are very rich in albumen, but in which one scarcely ever discovers a cast; while in some others, which, on boiling, exhibit the scantiest deposit of albumen, we find numbers of renal casts. An example of this last is seen in the secondary contraction which ensues after the subsidence of an inflammation of the kidney.

---

[1] Handbuch der systematischen Anatomie des Menschen. Bd. 2, S. 318.

According to my own experience and observation, I can still further extend the proposition laid down above, and state that *all those circumstances which give rise to albuminuria, may also originate cast formations;* and yet I am quite ready to admit that in not a few cases of albuminuria I have been unable, after repeatedly seeking for them, to discover any casts at all in the sediment. Still, in the larger proportion of cases of albuminuria thus tested—and their annual number is considerable—I have found the objects in question in the urine, no matter what may have been the cause of the albuminuria. Thus we find them in the albuminous urine of fever patients, when the albuminuria has lasted for but three days; they are not absent from the urine furnished by the cyanotic kidney of heart disease, so soon as the venous obstruction is enough to induce albuminuria; they are encountered in great numbers, both in acute and in chronic diffuse nephritis; they are present, though few in number, in the aqueous urine of simple contracting and amyloid kidneys; they are also present, finally, in cases of local deposits in the kidneys (for instance, hemorrhagic infarctions, directly the pressure causes the urine to become albuminous).

The observation of numerous instances of febrile albuminuria has taught me that the urinary casts and the albumen may appear in the urine at precisely the same time. Furthermore I have observed the same thing in two other cases—the one already quoted (Case III.), in which the albuminuria followed upon a blocking up of the ureter that had lasted for five days, and the following one:

CASE IV.—A journeyman mason, in November, 1869, fell a distance of thirty feet from a place where he was standing, and alighted on his buttocks. He was a little stunned, but shortly began to complain of sharp pain in his back, and had to be carried home. The first urine he passed, five hours after his accident, had a decidedly acid reaction, and deposited, on boiling, a flocculent precipitate of albumen; it further presented a slight dirty red-colored sediment, in which I found, besides red blood-cells and blood-casts, some perfectly homogeneous cylinders as translucent as glass, and pretty numerous. The next day's urine was brought me in so dirty a glass that it was useless to examine it; but in that which I examined a day later I could find no albumen, and could discover no casts at all in the sediment, which was loaded with numberless crystals of uric acid. The fellow got quickly and completely well.

Now this case appears to me to favor, in a very remarkable manner, the view which I have advocated above—and which is at the same time the view first held by the earlier authorities—namely, that the hyaline casts originate in the coagulation of some albuminous substance excreted together with the urine; and the following observation speaks scarcely less for the same opinion:

In May, 1874, a patient of mine underwent the operation of transfusion with lamb's blood, for splenic anæmia. His urine, which had been daily examined, up to the date of the operation, before this contained no albumen; while that which was passed two hours after it contained not only albumen, but some hyaline casts.[1]

It is pretty well known that many hours are not required for fibrine to coagulate out of the fibrine elements of the blood plasma. On the other hand, to assume that animal cells, situated within the body, perfect in themselves, and belonging to a sound organ, may, in so short a time as this, and under the influence of so slight a disturbance of the circulation, become converted into a homogeneous substance, in which no features of cells are any longer traceable, would be to assume something for which no analogue exists in the entire range of pathological experience. Quite equally contrary to experience would be the secretion of any substance of the kind described by the epithelial cells, following at so rapid a rate, and upon provocation itself so slight.

While now it must be conceded that albuminuria is an event which may be said to invariably precede the formation of true casts in the renal tubes—for the circumstance that in particular instances no albumen and some casts have been found cannot, for reasons already stated, be allowed to discredit the general rule; and while, further, it is established that albuminuria—be the source thereof what it may—is almost without exception attended by cast formations, it becomes quite impossible to resist the idea that some ingredient of the albuminous urine has a share in the formation of urinary casts. I further consider it as quite decided that the slight differences which have led authors to differentiate so many varieties of casts, are really brought about by metamorphoses which the casts experience between the time of their formation in the tubules, and the time when they are removed by the urinary secretion, changes whereby both their optical and their chemical properties are affected. I cannot think it other than likely that the urinary cylinders begin as homogeneous transparent casts; then, if they remain some length of time in the tubules, they acquire a fine granularity, and fat drops may form in their interiors—changes, so to speak, forced upon them by their age. In these changes brought about by their prolonged stay in the urinary tubules is probably to be sought the explanation of the fact established by Rovida, namely, that different kinds of casts conduct themselves quite differently with reagents, and present features notably distinct from those vouchsafed of strictly albuminous substances.

---

[1] See also *Huppert* in Virchow's Archiv. Bd. 59, S. 385.

It may well be a change produced by age, which, in rare examples, compels the waxy, shining, yellow casts to exhibit characters quite different from those which ordinarily appertain to these bodies; I refer particularly to the red-brown color which they assume upon the addition of a solution of iodine in iodide of potassium, and to the dark violet color which they afterwards assume when sulphuric acid is further added to the fluid containing them. This peculiarity I have witnessed in two cases. In both of them the actual quantity of casts contained in the sediment was excessive; and besides the shining waxy yellow casts, there were present numerous examples of both the white transparent and the dark granular cylinders in each drop of fluid examined. When I applied the iodine solution to the specimen under the microscope, the hyaline, colorless, and generally narrow cylinders acquired a yellow staining, just like that which is imparted to the precipitate of albumen when iodine is added to albuminous urine; while the thick, waxy, shining casts especially, and the dark granular casts in less degree, acquired a red-brown color, which was converted into a dirty violet upon further addition of sulphuric acid, the pale transparent cylinders, although colored yellow by the iodine, experiencing no further change. The violet coloration above noticed is only a very temporary affair, quickly changing into a dark black-brown. But exactly the same color reaction was displayed by some spherical bodies or irregular-shaped flakes composed of some homogeneous substance and presenting the same shining appearance and the same color as did the waxy casts themselves. In the second case observed by me, I found enormous numbers of these bodies in the sediment, in addition to casts of great variety. In the sediment, from the first case, I am not prepared to say positively that the bodies were spherical, for I have no record to this effect in my notes. When I first observed them again in my second case, I took them for degenerated and enormously swollen epithelial cells, although I could make out no trace of a nucleus in their interiors; in fact, it was only in certain of these spheroids, and after coloring them by the iodine solution, that I was able to make out a narrow pale portion surrounding like a border a darker colored central portion. Still my present idea is, that both these spheroids and the flakes consisted of one and the same substance as that of which the waxy casts excreted by the same kidneys were formed, and that both are a species of secretion proceeding from the epithelial cells of the tubules, and deserve to be regarded as smaller forms, evidencing an identical causation.

CASE V.—In only one out of these two cases (namely, in the second) was I able to inform myself as to the state of the kidneys, by examining the body of the person in whose urine during his life I had observed these objects. He was a phthisical patient, aged forty-two, brought into my Clinique with swollen and highly œdematous lower extremities, on October 30, 1863. He was nearly voiceless from an ulcerative destruction of the vocal cords, and had had diarrhœa for some time. He secreted very small quantities of urine, from 500 to 700 c. ctm. in the twenty-four hours, of a specific gravity varying from 1007 to 1010 on different days. In one day's urine the albumen measured 0.975 per cent. There were enormous quantities of the above-described casts constantly in the sediment. He died of hæmoptysis on the 29th November. The autopsy discovered numerous large and small cavities and numberless deposits of miliary tubercles in the lungs. The spleen was twice the size it ought to be. The liver was considerably enlarged and adherent to the diaphragm; its edge was rounded in a peculiar mannner. Both kidneys were much enlarged, and in a condition of amyloid degeneration; the capsular vessels were greatly distended; the entire cortex appeared spotty upon section, yellowish-gray colored and shining; the medullary portion was of firm consistence, rather fuller of blood than normal. Thin sections taken from the cortex were transparent, and gave a distinct amyloid reaction with the iodine solution. Unfortunately no microscopic examination was made.

No doubt the other case was of the same kind, for it occurred in the subject of an atonic ulceration of the leg of enormous size and long duration; but the man withdrew himself from my observation before he died. Still I do not think one is justified at once in making a diagnosis of amyloid disease of the kidney in every case because the above-described kind of casts have been discovered, nor in excluding the possible existence of such disease when cylinders, which do not give this special reaction, are forthcoming. Both these cases came under my care in the years 1859 and 1863. Since then I have had under observation many cases of renal amyloid disease, and have paid special attention to the casts presented, testing them with iodine; but I have come across no further instance of the characteristic reaction.

Now, just as in the two cases mentioned above, it was not all the broad waxy cylinders which exhibited the peculiar color reaction, so it appears to me to be highly probable that a metamorphosis into amyloid substance takes place only after some lapse of time, and in those casts which stay a long while in their birthplace, and are not swept away into the urine directly they are formed. Friedreich[1] showed long since that old-standing fibrinous sediments, in the interior of hæmatoceles, suffered an amyloid metamorphosis. Why then should we refuse to accept the possibility of an albuminous substance experiencing a like conversion in the tubuli uriniferi, after it has been retained a long time inside them? It is true enough, as experience teaches us, that this conversion is a rare one, that we do not know the conditions requisite to effect it, and that we are ignorant whether or not the amyloid degeneration of the renal vessels may exert an influence upon this change, and if so, what is the nature of that influence. I must here, however, state explicitly that, in both the cases I have cited, my observations extended over a considerable period of time, in one case, for instance, over six months; that, owing to the great interest which was excited in my mind by this peculiar phenomenon, I examined the urinary sediment nearly every day; and that the casts which presented this characteristic reaction with iodine were found with the greatest constancy.

---

[1] *Dr. N. Friedreich* and *Dr. A. Kekulé*, Zur Amyloidfrage. Virchow's Archiv. Bd. 16, S. 50.

But I would make mention here of another case of amyloid degeneration of the kidneys—although the case itself must stand over, for fuller description, to a later part of this work—in which the cylindrical casts themselves only assumed a yellow coloration upon addition of the iodine solution; whereas renal epithelial cells, some of them singly, others hanging together, others again clinging to the hyaline casts, became bright red, and then purple when sulphuric acid was further added. Indeed the contrast of coloration was of a most striking kind in the cells which clung to the casts.

Now, what interpretation are we to attach to the presence of casts in the urine? Certainly no more than this, that the person who passes them is suffering with true renal albuminuria. But the presence of casts themselves does not, in the slightest degree, appoint the nature of the cause at work in producing the albuminuria. At the same time the value of the characters of casts in assisting us to make a diagnosis is very great.

If the quantity of casts present in the sediment be reckoned alongside of the quality of the urine, and due weight be given to all other attendant circumstances, inferences may be drawn as to the nature and extent of the disorder to which these bodies owe their origin. As previously noticed, we meet with some albuminous urines in which we must search a long while to discover a single cast, and others, again, which deposit a copious sediment consisting of almost nothing else but casts.

A urine which contains a great number of pale or dark granular casts comes from an inflamed kidney. If a great number of undamaged epithelial cells from the tubuli uriniferi cleave to these pale casts, and there is a fair abundance of red or white blood corpuscles, and few or no dark granular casts are present in the sediment, the case is evidently one of acute nephritis. If, on the other hand, dark granular, and oftentimes puckered, casts prevail over the pale and hyaline ones, the case is then one of chronic nephritis.

The urine of febrile and congestive albuminuria, as well as that of contracting kidneys, and of most examples of amyloid degeneration, presents only very few casts.

The shorter the time an albuminuria has lasted, the more likely one is to find, besides a few true epithelial and blood-casts only, perfectly pale homogeneous and usually narrow cylinders; this is the case, for example, in febrile albuminuria. But even in most cases of chronic albuminuria, in which a persistently large amount of watery urine is secreted, the narrow pale casts usually preponderate over the thick and dark ones; it is so, for instance, in genuine contracting kidney and in the larger proportion of cases of amyloid degeneration of the kidneys. Another matter, which must be taken into consideration in these cases, is the rapidity with which the secretion of urine is proceeding. Thus the urine, which is secreted under circumstances of high blood pressure, flows in a swift current, and sweeps out all cylinders, which may chance to be in the urinary tubules, before degenerative changes of any advanced nature, such as time could effect, can have taken place in them; and in the straight and wide tubules of the pyramids, through which the whole flush of fluid flowing from the cortex has to pass, the conditions will at least be most unfavorable for either the formation or the retention of casts. When, therefore, we find a great many broad cylinders in the urine, we may be pretty sure that we are dealing with a condition of the kidneys in which the secreting process is obstructed, as is the case, for example, in acute nephritis and in some instances of amyloid degeneration. Indeed I have found the largest number of thick-sized casts, some of them dark granular and others waxy, in the secondary contraction of the kidney which follows after chronic nephritis, and in some examples of amyloid disease of the kidneys. In all these cases the quantity of urine secreted was invariably small, and, in spite of its small quantity, was of a pale color, and of a low specific gravity, and contained, besides a few small pale casts, large cylinders, as a rule in such numbers as to constitute a considerable layer at the bottom of the vessel holding them, forming a white, dust-like sediment. Under such circumstances the speedy termination of the case can be confidently expected. For the presence of these broad casts in large numbers in the urine with certainty indicates that a large portion of the kidney has suspended work, and one can confidently affirm that the epithelium of the kidney is correspondingly degenerated, and that casts have not merely been formed, but have remained a long time in the straight excretory tubules of the pyramids.

It has been maintained that the waxy, refracting, and oftentimes yellow-colored casts, are only forthcoming in amyloid degeneration. This is not correct, for I have found them in the sediment from a case of chronic nephritis, and satisfied myself, after the death of the patient, that the large fawn-colored kidney had not suffered the least amyloid degeneration. My late colleague, Colberg, in a case of this kind, which came from my Clinic, found that nearly all the straight tubules were blocked up with these waxy casts. (Compare the appearances seen in the drawing, Fig. 7, which is taken from a preparation made from this kidney.)

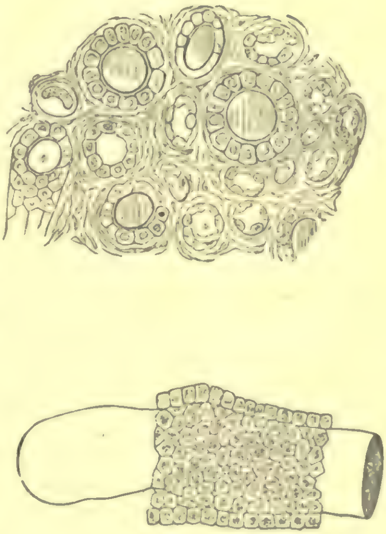


FIG. 7.

Waxy refracting casts in the interiors of tubules, which still retain their epithelial lining. (Colberg.)

This much only can one say, namely, that the waxy casts always appoint a chronic and deep-seated renal affection, and are never present in recent cases of nephritis nor in transitory albuminuria. Further, the dark granular casts never appear in the temporary functional disorders of the kidneys ; they indicate a notable impairment of the nutrition of the organ. At the same time their presence does not justify any deductions regarding the nature of this mal-nutrition, as they are found both in nephritis and in amyloid disease of the kidneys.

Narrow hyaline colorless casts can appear in any albuminous urine. They continue as long as the albuminuria lasts, and, if carefully looked for, they are always to be found in company with the dark granular and waxy cylinders, although often only here and there a specimen.

Whenever waxy casts are present in the urine, one generally finds all three forms of casts at the same time in the sediment.

In very rare examples the casts shed in disease of the kidney are themselves so numerous as to bestow a dark citron-yellow coloration to the fluid, which, at the autopsy, is found in the pelvis and calices of the kidney.[1]

III. The Disorders manifested by the Blood, by the General Nutrition, and especially by the Nervous System, when the Kidneys cease to perform their Functions properly.

We advance, then, to the disorders that arise from the malperformance of their natural functions by the kidneys; and the natural functions, whose failure we have to consider, are those previously pointed out by us—namely, the regulation of the watery content of the blood serum, and the excretion of the nitrogenous waste resulting from the nutritional interchanges.

If we take the first—the results of an insufficient removal of water from the blood—we find ourselves face to face with

1. *The Dropsy that accompanies Renal Diseases.*

Hippocrates already discovered that the diminution of the urinary secretion was a cause of dropsy, and established the source of particular forms of dropsy in the loins (the kidneys).[2] Van Helmont described the kidneys as the principal originators of dropsies. Still the connection, as cause and effect, between kidney disease and dropsy was not generally understood until Bright's work upon this subject first cleared the way.

---

[1] *Prof. Ackermann*, Ein Fall von Parenchymatöser Nephritis mit Retention der Cylinder in den Nierenkelchen und im Nierenbecken. Deutsches Archiv für klin. Medic. Bd. 10, S. 298.

[2] Hippocratis opera. Ed. Fœsius. Francofort, 1624, p. 190.

From this time on no one disputed the correctness of the statement that the dropsy of renal disease was due immediately to hydræmia, an abnormal increase of the watery content of the blood serum; a fact, too, confirmed, since Bright's time, by no small number of analyses made upon the blood of patients thus afflicted. Bostock, a friend of Bright, and one who accomplished for him the accurate analysis of the urine in the cases contained in his paper, found that the blood serum of one of these patients presented a specific gravity as low as that exhibited by the urine itself, namely, 1013[1]—the very lowest specific gravity for blood serum which he had ever seen. The patient from whom both the urine and the serum were derived was dropsical, and passed exceedingly little urine. The coagulum produced by heating the blood serum was hardly so great as that obtained from the urine when this was boiled.

Christison, who reckoned 1029 to 1031 to be the normal specific gravity of the blood serum, seldom found that that derived from the subjects of renal dropsy presented a higher average than 1022, and noticed one case in which it stood at 1019. He found that the solid residue of the serum in one case of renal disease amounted to only 61 parts per 1000; whereas, normally, they should reach to 82 or 85 per 1000. The results of some examinations, which I instituted myself upon the same subject, would be expressed by the figures 1018, 1016, and 1015, as the average specific gravity of the blood serum in renal dropsy, and similar observations, of which numbers exist, made by Rayer and other authors, pointed to quite the same conclusion.

In regard to the real cause of this hydræmia, and therefore of the dropsy, the views of many physicians and medical writers at the present time do not appear to me to be quite clear. It is certainly correct that those forms of renal disease are most apt to induce dropsy which are associated with the escape of large quantities of albumen in the urine, and the fact admits of no dispute that the persistent excretion of albumen from the blood serum in this abnormal manner must render the latter more watery. Wherefore the opinion of those who attribute the dropsy of renal disease entirely to the waste or loss of albumen in the albuminuria is not at once to be rejected; only this loss of albumen is in most cases neither the sole nor the essential cause of the hydræmia and dropsy, and in a good many instances it may be left out of consideration altogether.

---

[1] *R. Bright's* Report of Clinical Cases. 1827, Vol. 1, p. 85.

In evidence of this being the case, I might cite the fact that renal disease can lead to most extensive dropsy, when, throughout its course, no quantity of albumen to merit mention has ever escaped in the urine. For instance, dropsy may ensue within a very few days in the acute nephritis accompanying scarlet fever or diphtheria, the urinary secretion coming to an absolute standstill, and complete suppression of urine being established; and these cases prove beyond dispute that the non-removal of water from the blood by the kidneys, while the absorption of water by the digestive tract still goes on as in health, can be the source of the hydræmia and dropsy. But even in other cases of renal disease, the occurrence of dropsy depends less upon the amount of albumen actually lost in the urine than upon the diminution of the amount of water excreted by the kidneys.

The cases of dropsy occurring in heart disease especially confirm us in this idea. Still even for these cases the explanation has been offered that the dropsy is due to the impoverishment of the blood serum in solids, and especially in albumen, which results from faulty assimilation produced by disordered digestion. At the same time the advocates of this view have felt obliged to attribute some influence to the increased blood pressure in the veins. It seems to have been forgotten that there are diseases of the digestive organs which induce far graver states of marasmus than usually attend on heart disease, and yet do not produce any dropsy. In the study of this question, however, far too little importance has been attached to the diminution of the amount of water secreted by the kidneys, which follows as the result of decided valvular defects of the heart. It is only when, in consequence of insufficient cardiac energy, the arterial blood pressure falls so low that the renal secretion is palpably diminished, that dropsical swellings commence, and then subside again directly the urine flow is accelerated by the heart's action becoming once more vigorous.

Urinary excretion and dropsy, in patients suffering from heart disease, undoubtedly stand in direct antithesis to each other, the latter decreasing as the former increases, and vice versa. The paltry loss of albumen which takes place through the kidneys does not merit notice in regard to its bearings upon the dropsy. But it is correct enough that the local extension of dropsical effusions in heart disease is determined by the differing degrees of blood pressure ordinarily experienced in different sections of the venous system.

Just the same dependence of the dropsy upon the quantity of the urinary water secreted, may be observed in renal diseases proper. Only those forms of kidney disease in which an abnormal diminution of the urinary secretion takes place, give rise to dropsy with any certainty. In most cases, it is true, the diminution of the renal secretion is contemporaneous with the appearance of albumen in the urine, and it is certainly correct that an abundant loss of albumen of the blood by other channels, as by a hemorrhage, may be followed by dropsy. Only this nowise diminishes the importance of the fact, that the advent of dropsy in renal disease is directly dependent on the lessened secretion of urine and not upon the quantity of albumen excreted in that urine. For a considerable loss of albumen may take place in this way without any dropsy occurring if only the quantity of urine passed diurnally is sufficiently copious, and dropsy may ensue during an insignificant loss of albumen directly the daily urinary secretion falls below a certain minimum quantity. The dropsy produced by kidney disease may disappear, notwithstanding the fact that the loss of albumen remains each day the same, if only the quantity of water excreted by the kidneys becomes greater.

Dr. J. Rehder, with the view of approximately establishing the proportion borne by the quantity of water taken in drink and food towards that which afterwards appeared in the urine, instituted a series of investigations upon sound, healthy persons who were living under identical external conditions, and then compared the results with a series of analogous observations made on dropsical subjects. He kept all these people, the healthy as well as the sick, in bed throughout the period over which the investigation extended. Nearly all the experiments were made in the cold season of the year.

The quantity which could be reckoned as fluid nourishment, drink, or soup, was, in the case of each person experimented with, most strictly measured, while the water contained in the solid food was left out of consideration. Then the entire urinary secretion, passed through the period of observation, was collected together, and its quantity measured and compared against the quantity of the ingested fluids.

Of the five healthy persons (that is, who had no renal malady), serving for the first series of experiments, two had gonorrhœa (robust young men), and three suffered with spondylitis (well-nourished and fine-looking young girls, aged between sixteen and twenty-three).

Now as regards these five persons, two of whom were kept under observation for four days, and three of them eight days, or twice as long, the results were as follows: of every hundred parts of fluid taken internally sixty-eight, seventy, seventy-six, eighty, and eighty-eight parts respectively, came to light again as urine. But it appeared that these figures were subject to enormous variation upon single days, the consequence, no doubt, of the irregularity with which the urinary secretion is effected in the different individuals. The following table exhibits the amount of variation:

						Per cent.			*Mean average per cent.*
Individual	I.	Observation	extending	over	8 days.	Between 20.9	and	250	70
"	II.	"	"	"	"	62.5	"	109	80
"	III.	"	"	"	"	47.2	"	90	68
"	IV.	"	"	4	"	40.0	"	135.6	88
"	V.	"	"	"	"	52.2	"	111.7	76

Upon an average, then, of the five persons together, and the thirty-two days' observations, we may assume that, of every 100 parts of fluid ingested, 76.4 parts are excreted in the urine.

Very contrary results were arrived at by the observations, upon the relation of the fluids ingested to those excreted, made upon persons who were suffering with cardiac or renal dropsy.

A man, fifty years of age, who had had long-standing aortic insufficiency, and had shown all the symptoms of progressive cardiac enfeeblement, cyanosis, advancing œdema of his connective tissues, etc., took, during five successive days, 6,782 c. ctms. of fluids, in the form of drink and nourishment, together making a daily average of 1,356 c. ctms.; during the same time he excreted altogether 2,645 c. ctms. of urine, or a daily average of 529 c. ctms. That is, for each 100 parts of water ingested by him, 39 were excreted by the kidneys, the variation exhibited upon individual days fluctuating between 29.7 and 49.2 per cent.

A youth, St——, sixteen years of age, afflicted with chronic parenchymatous nephritis and increasing general dropsy, took, during five successive days, 5,350 c. ctms. of fluid, or a daily average of 1,070 c. ctms., and excreted during the same period 1,330 c. ctms. of urine, making a daily average of 266 c. ctms. The relation, therefore, of the fluid ingested to that excreted was in the proportion of 100 : 24.86, and the variation upon individual days ranged between 18.6 and 33.5 per cent.

The next case was that of a woman, aged forty, who had chronic parenchymatous nephritis, complicated by amyloid degeneration; she became dropsical, and died, on December 8, 1873, with most extensive general anasarca and ascites. This woman took, in six successive days (that is, between the 13th and the 18th of November), 7,020 c. ctms. of fluid—upon an average, therefore, 1,170 c. ctms. daily; and she excreted during this time 1,765 c. ctms. of urine, or an average of 294 c. ctms. daily. The relation, therefore, of the fluid ingested to that excreted through the kidneys is as 100 : 25, and the variation upon individual days ranged between 10.8 and 40.2 per cent. The experiment with her was repeated after an interval of ten days, during which time the dropsical symptoms had increased. From the 28th of November up to the 5th December she took 9,360 c. ctms., or a daily average of 1,170 c. ctms., and excreted in the same period 1,495 c. ctms., or a daily average of 187 c. ctms. of urine. Thus the fluid ingested stood in proportion to that excreted by the kidneys in the proportion of 100 : 16, and the variation in quantity upon particular days ranged between 11.5 per cent. and 24.4 per cent.

An engineer, thirty years of age, the subject of chronic parenchymatous nephritis, which succeeded malarial poisoning, a man who interested himself in the result of the experiments, and whose intelligence made me confident that they would be thoroughly carried out, took, on fifty-two succeeding days, between February 17th and April 10th, 1874, 171,150 c. ctms. of fluid, and excreted during the same period 48,840 c. ctms. of urine, the daily average, therefore, standing thus: 3,291 c. ctms. of ingested fluid and 920 c. ctms. of urine—a ratio of 100 : 28.

Now, this patient, whose whole body was swollen and altered in shape by his anasarca, took a hot air bath every day throughout this period, and therefore perspired profusely; and still his dropsy resisted the treatment.

For purposes of comparison, I now subjected a woman of forty (who, beyond having chronic rheumatoid arthritis (arthritis deformans), was otherwise sound) to a sweating or hot-air cure, while I experimented upon the fluids ingested and excreted by her. In nine successive days this woman took 13,580 c. ctms., of fluids, or a daily average of 1,510 c. ctms., and excreted a total quantity of 5,800 c. ctms. of urine, an average of 645 c. ctms. daily, or a proportion of 100 : 42.7.

The following case has especial interest, for it shows in the clearest manner the dependence of renal dropsy upon the quantity of urine excreted by the kidneys. B., a laborer, thirty-four years of age, suffered with dropsy and general anasarca in variable degrees, in consequence of parenchymatous nephritis. He was treated with hot-air baths. When Dr. Rehder began his observations upon him the dropsy was on the decrease. During the first five days that he was experimented upon, between the 8th and the 12th of July, 1873, he took 7,565 c. ctms. of fluid, and excreted 7,600 c. ctms. of urine, taking therefore less water than he passed as urine; the ratio being as 100 : 100.46. His dropsy now increased again. Between the 13th and the 15th of July the quantity of water ingested amounted to 5,575 c. ctms.; the quantity of urine excreted amounted to 1,858 c. ctms. The ratio was as 100 : 33.3.

From the 16th to the 20th of July the dropsy was again on the decrease, and the ratio of fluid ingested to that excreted was as 100 : 98.3.

From the 23d to the 27th of July, increase of swelling again. Ratio ascertained to be as 100 : 48.8.

From the 23d to the 26th of August, considerable œdema of the face. The fluids ingested stood to the urine excreted as 100 : 29.

From the 29th August to the 3d September, œdema of the face abating. Ratio of ingested to excreted fluids as 100 : 72.5.

In September this patient's state remained unaltered, and the fluid ingested stood to the urine excreted during four successive days as 100 : 65.2; and in October, the dropsy being again on the increase, the ingested fluids stood to the urine excreted in the proportion of 100 : 32.6 for four successive days.

These experiments, leaving as they do much to be desired in accuracy, still taken together are convincing evidence that the dropsy in renal disease is due to the altered elimination of water by the kidneys, or, to speak more exactly, to the relative insufficiency of these organs for this part of the task which they have to fulfil—an insufficiency which establishes a non-correspondence between the ingestion and the excretion of water. Hitherto I have not had the time to make these experiments upon a larger scale and according to a more exact method, the pressure under which I have had to do my work not allowing me the requisite leisure.

It follows, then, from what has been stated, that the renal diseases associated with albumen in the urine are not the principal ones which lead to dropsy; other forms of kidney disease are more likely to produce this, and especially all such as influence the quantity of urine excreted, and can compel complete suppression of urine, whether this be due to changes in the kidneys themselves or to blocking up of the excretory channels leading from them. I will not, however, leave the fact unmentioned here that even complete arrest of the urinary secretion must last a certain time before dropsy follows it. In one of the cases which I have narrated (Case III.), where suppression followed the stoppage of both ureters, 122 hours elapsed without a trace of œdema showing itself. Still, I must also say that from the beginning this patient suffered with most serious vomiting, and rejected by the mouth large quantities of fluid—chiefly drinks, it is true, which had been taken freely.

A case of similarly protracted complete suppression was observed by Biermer, in nephritis after scarlet fever, and in this, too, the previously existing dropsy scarcely increased at all throughout the five days' absence of all urinary excretion.

In far the larger proportion of cases of renal dropsy it is not a destruction of those organs which are specially destined to secrete the urinary water—the Malpighian bodies—or the resulting diminution of their number, which leads to lessening of the secretion and so to dropsy, but the obstacles which are opposed to the free discharge of the secretion through the renal tubules. Quite one-half of the urine-preparing apparatus can be wanting without the other half being insufficient to accomplish its physiological task, as numberless cases of congenital deficiency and of the entire destruction of one kidney, which we find recorded, testify. Indeed, as Simon's bold and successful operation proved, even the sudden removal of a normally functioning kidney, from an adult in the fullness of his strength, can be supported, the remaining organ sufficing to remove the necessary quantity of water and other urinary solids from the blood. Wherefore, even in the diffuse affections of the parenchyma of the kidney, it is not the contracting forms, associated with destruction of numberless Malpighian bodies, which we find preferentially producing dropsy. It is not uncommon at an autopsy to find the kidneys wasted away to mere degenerate relics, in persons who, during life, exhibited no trace of dropsical anasarca.

On the other hand, dropsy is scarcely ever absent in those acute and chronic processes of inflammation of the kidneys in which these organs, through swelling of the epithelium and of the interstitial tissues, have considerably increased in size; for this swelling presents a considerable obstacle to the normal secretion pressure within the Malpighian bodies, and in this way obstructs the excretion—a circumstance only possible while there is considerable difference between the blood pressure within the glomeruli and that offered by the fluid contents of the tubuli uriniferi.

It follows of itself that dropsy must ensue whenever there is a protracted diminution of secretion pressure in the secreting vessels—as happens in heart disease—and all the more easily if the actual number of the secreting vessels themselves is lessened. Wherefore dropsy is wont to be the last event in the course of progressive contraction of the kidney, when, in consequence of this, the general nutrition, and so the vigor of the heart itself, is impaired. But so long as in this common kidney affection the energy of the heart as a pump, is maintained, and the excessive diuresis, commonly accompanying it, continues, dropsy does not make its appearance, although the albuminuria may have endured for years.

Dr. Grainger Stewart[1] has promulgated views entirely in accordance with my own upon the essential cause of dropsy in kidney disease. He allows that the dropsy in nephritis may be attributed to the diminution of the serum albumen ; but he does not accept this as the sole reason ; since the dropsy often forms the first symptom of the disease, and is more apparent early in the course of the inflammation than in its later stages, although the blood then, from protracted loss of albumen, must have become very much more deteriorated. The dropsy, he continues, must surely rather be due to the non-elimination of water through the kidneys, and to the resulting increase of the blood pressure upon the walls of the capillaries and veins. The less the urine secretion is, the acuter and more copious will be the dropsy, and the latter will subside directly the water can again flow off by its natural channels.

The nature of the transuded fluid, which in renal dropsy passes through the walls of the capillaries into the subcutaneous connective tissue, and which accumulates in the serous cavities of the body, expresses in a most marked degree the extreme impoverishment which the blood serum has undergone in so many of these cases, through the retention of water in the system. The dropsical fluid is a filtration merely of the blood serum through the walls of the vessels, and it constitutes a watery solution of salts exceedingly poor in albuminates. Its actual solid contents will be all the scantier, the more watery the blood serum is from which the transudation came, for the albumen content of the dropsical fluid is regulated exactly by the albumen content of the blood serum.

---

[1] A Practical Treatise on Bright's Diseases of the Kidneys. Second Edition, Edinburgh, 1871, p. 83.

The aspect of this fluid is somewhat variable. That obtained from section of the connective tissue is generally as transparent as water; that taken from any of the serous cavities of the body is, on the contrary, as a rule, pale yellow or light-green colored, and fine, thread-like shreds or coagula are found in the latter, while the fluid itself is slightly opalescent, from the epithelial débris mixed with it.

The composition of the dropsical fluid varies, however, in the dead body, according to the locality from which it comes. That which is poorest in solid constituents and in albumen is pretty invariably the fluid furnished from the subcutaneous connective tissue. C. Schmidt found that, in a case of renal disease, the fluid contained in the pleura gave 2.85 per cent. of albumen; that derived from the peritoneal cavity, 1.13 per cent.; the meningeal fluid from 0.6 to 0.8 per cent.; and that from the subcutaneous tissue of the skin, 0.36 per cent.

To exhibit these differences at a glance, I have arranged, in the form of a table, some analyses of the blood serum, of the fluids derived from the pericardium and the peritoneum, and of that obtained in anasarca generally, all of which were taken, directly after death, from the body of a person who had died of advanced dropsy.

	Specific Gravity.	Water.	Solid Constituents.	Inorganic Salts.	Albumen.
Blood serum................	1015.58	957.656	41.402	?	30.39
Pericardial fluid............	1009.69	978.62	21.38	15.58	?
Peritoneal fluid.............	1009.63	984.31	15.69	11.48	2.55
Anasarca fluid..............	1007.65	988.30	11.70	10.69	Traces.

I have frequently found extremely small quantities of albumen in the fluid which oozes from the swollen extremities, in renal dropsy, after the epidermal layer has burst. But I have never been able to discover urea in the fluid of this anasarca, although my colleague, Edlefsen, once found 0.359 per cent. of urea in it, the blood, taken from the same body, giving only 0.258 per cent. of this same substance. Edlefsen once found 0.28 per cent. of urea, and another time 0.30 per cent., in ascitic fluid ; and he was able, in another case, to show the presence of an entire 1 per cent. of urea in the pericardial fluid. In this last instance, however, the blood serum was so loaded with urea that this had passed over in quantities into the secretion of the sweat glands, and, after the evaporation of the water, had remained in the form of crystals upon the surface of the skin of the face and neck. We were able to scrape off and collect from the skin of this individual, while he was alive, considerable quantities of urea crystals.[1]

The distribution of the dropsical exhalations into different organs and over different parts of the body is quite peculiar to renal dropsy, and departs from the order which holds good in dropsy from other causes. The law which we observe in renal dropsy is for the accumulations to take place, first and foremost, in the subcutaneous cellular tissues. It begins as anasarca, and, with rare exceptions, the dropsical effusions proceed next into the serous cavities, and subsequently invade the alveolar tissue of the lungs and the submucous cellular tissues.

General anasarca may exist for a long time before dropsy invades one or more of the great serous cavities of the body. It depends upon the external conditions in which a patient is placed—that is, whether he lies in bed or moves about, when the dropsy is beginning—what particular part of his body will first show signs of becoming œdematous. With some patients the first thing noticed is a slight œdema of their ankles, noticed when they undress in the evening, at the end of their day's work ; with others it is a swelling of the eyelids, which has taken place during the night, and remains when they get up in the morning. More rarely it happens that the first swelling observed shows itself in some other parts of the skin, which present a loose and wide-meshed subcutaneous cellular tissue—as, for instance, the præputium or scrotum in men, labia pudenda in women.

---

[1] *Dr. Henning von Kaup* and *Dr. Thos. Juergensen*, Ueber Harnstoffausscheidung auf der äusseren Haut beim Lebenden. Deutsches Archiv für klin. Med. Bd. 6, S. 55.

At first, and while the dropsy is still slight, in renal cases, these swellings exhibit a remarkable fugitiveness and inclination to change their locality. The same individual, whose eyelids, on getting up in the morning, were so swollen as to completely close his eyes, at night, after being about all day, opens his eyes with ease, but finds his feet are swelled ; or, lying in bed and sleeping on one side, that side of the face on which he lay will, upon his waking, be considerably swollen, the other being scarcely affected at all, and the whole condition will be reversed again upon his altering his position in bed. The influence of gravitation evidently plays a part here, as do other matters, which are not in every instance capable of being discovered, any more than are the reasons why, in particular examples of renal disease, the œdema should be entirely circumscribed, or present itself in quite unwonted places, and remain in them sometimes with remarkable persistence.

The parts of the body which are swollen and anasarcous, in consequence of renal disease, are invariably strikingly pale, and the skin over them is, as a rule, quite remarkably dry and, in extensive dropsy, so stretched as to be, together with the fluid within it, quite transparent to transmitted light. In fact, the distention may lead to such a loosening of the connective tissue of the skin, that strips of this are thinned, and, after subsidence of the swelling, may present themselves as white, scar-like streaks, exactly similar to the lineæ albicantes upon the abdominal walls which follow pregnancy, and owe their origin to the self-same stretching. Then the epidermis itself may be torn, and thus give opportunity for the dropsical exudation to trickle away.

The swelling of the entire body, produced by the hydrops anasarca of renal disease, sometimes reaches to an extreme degree ; and these are the cases in which the dropsy is of a quite unusually stubborn kind. It may last for years ; but the graver forms of dropsy are generally sooner or later complicated by effusions into the serous cavities, or by lung œdema, œdema of the glottis, or of the mucous membrane of the stomach and intestines. And yet there may be a considerable anasarca, lasting a long time without any one of these complications.

According to my own experience, it rarely happens that the accumulation of fluid into one or other serous cavity has been the commencement of the dropsical symptoms. More often I have observed acute lung œdema as a first symptom of what was then shortly a fatal dropsy. Œdema of the glottis I have never seen as an initial symptom of renal dropsy, although other writers have—indeed, œdema glottidis has happened in exceedingly few of my cases of renal disease.

Next in order of consideration come the consequences of an imperfect depuration of the blood in renal disease, the results of the retention in the blood of the dross of the capillary interchanges, the nitrogenous substances and specific urine constituents.

2. *Uræmia.*

Under this name a series of symptoms have been reckoned together, which have been held to be the direct result of blood poisoning with urinary matters, although, as time has gone on, very different interpretations have been given to them.

It is the general custom to regard as symptoms of uræmia very various functional disorders of the nervous system, which at times pursue an acute, at others a chronic course.

Acute uræmia, in the acute as well as in the chronic diseases of the kidneys, manifests itself in the form of epileptic convulsions, which are often terribly violent, and are succeeded by coma, or, in some instances, by a condition of maniacal excitement. In the larger proportion of cases the convulsions succeed each other at short intervals, oftentimes before the patient can recover from the coma of the preceding seizure. Not unfrequently a series of such epileptic attacks terminate at last with death. Cases, however, occur in which patients afflicted with a chronic renal affection, who have had one or a few such attacks following acute uræmic poisoning, recover again completely, and live on for a long time, pursuing their wonted avocations. Then, occasionally, epileptiform convulsions are the first symptoms to suggest an insidiously progressing renal disease, attacking, as they do, an individual in apparently perfect health. But in other cases these acute uræmic fits are preceded by other striking symptoms of the complaint—dropsy, dyspepsia, obstinate vomiting, etc. In rarer instances the characteristic epileptic convulsions are ushered in by cramp-like twitchings of particular groups of muscles, lasting for some days, or by tetanic spasms, which at first do not disturb the intellectual faculties. But far more commonly the first epileptic seizure assails the patient quite on a sudden and without any of these premonitions.

Amaurosis uræmica is one of these acute, suddenly appearing symptoms—though subsiding as a rule as suddenly as it comes—which has been reckoned amongst the group of uræmic symptoms. One must, however, distinguish this sudden and complete blindness from those disturbances of vision which so often occur in the course of chronic renal disease and which are due to gross structural alterations of the retina, alterations which are permanent in character. The ophthalmoscopic examination of the eyes of persons, who while suffering from kidney disease have become thus suddenly amaurotic, has, so far as I know, afforded no satisfactory explanation of the immediate cause of the blindness; its etiology is certainly not to be sought for in any great pathological alteration of the visual apparatus, though it may lie, perhaps, as Dr. Crocq[1] supposed, in simple œdema of the retina.

Convulsive phenomena may be entirely absent in *the chronic form of uræmia*, or they may appear only in the shape of twitchings of certain groups of muscles. The uræmia may show itself only by increasing somnolence, apathy, or stupefaction, advancing at last to complete coma. But this mental obfuscation is very often preceded by dyspeptic difficulties, by stubborn and incessant vomiting, and the vomited matters in such cases quite often contain large quantities of carbonate of ammonia, which gives it an alkaline reaction, can be perceived by the smell, and admits of chemical recognition without any great difficulty. Often enough in this form of uræmia there is a most tormenting itching of the skin, which drives the patient, though in a stupefied state, to continually scratch himself. Lastly, it may happen that a patient, who has been lying some days in a state of complete coma, is seized by one or by repeated epileptic attacks before death closes the scene of his sufferings. Further, one must reckon amongst uræmic symptoms those asthmatic attacks which sometimes assail the subjects of chronic renal affections a long while before their death. These attacks of asthma, like all neurotic asthmas, come on in paroxysms, with intervals of complete freedom, and are most frequent at night-time. I must defend the uræmic origin of these attacks against the opinion which Rosenstein entertains as to their nature. I have observed them in individuals in whose respiratory and circulatory organs no other explicable cause for such exceedingly violent fits could be discovered than bronchial spasm; and the real cause of this spasm, I believe, was uræmia.

---

[1] Presse medic. belge. October, 1850, p. 393.

The relation of these neuroses to renal disease was already recognized by Bright, although among his contemporaries different opinions were entertained as to their immediate cause. Christison on the one hand, attributed the suspension of the cerebral functions to granular degeneration of the kidneys, which, as he said, poisoned the blood by allowing the urea to accumulate in it, and impoverished it by robbing it of its coloring matter. Osborne, on the other hand, thought he perceived the real source of the convulsions and of the coma in an arachnitis, which he considered sufficiently identified by the presence, post mortem, of a slight opacity of the arachnoid membrane. And this division of opinion has, with slight modifications, come down even to the present day. Little by little, however, the theory of the contamination of the blood with urine elements and the poisonous effects of this upon the nervous system gained the upper hand. The idea was chiefly fostered by the proofs, first furnished by English observers, of an accumulation of urea in the blood of many patients affected with kidney disease. Babington[1] declared that he found during life 1.5 per cent. of urea in the blood serum of a woman, twenty-four years of age, who was dropsical, had albuminous urine, and finally died with epileptic convulsions, the autopsy showing her to have had granular kidneys; and he remarked on it that this woman's blood serum was as rich in urea as her urine.

In the meantime Owen Rees[2] raised some objection to the view that accumulation of even large quantities of urea in the system could be the cause of those severe and frequent cerebral symptoms that were presented in renal disease, and in favor of this view he cited a case which he had observed, in which, while one kidney was altogether absent, the ureter of the other, enlarged by compensative hypertrophy, had been blocked up by a calculus. The consequence had been complete suppression of urine. For all which, the patient had remained in complete possession of his mental faculties up to the last moment; and still Owen Rees found more urea in the blood drawn from this patient during life than had ever been shown to be present in the urine of any case of Bright's disease known to him. The results of experiments instituted upon animals also do not lend support to the doctrine, the injections of filtered urine and of solutions of urea failing to induce fits or conditions at all like uræmia.

Nevertheless Frerichs adopted this theory in a somewhat modified form.[3] At page 107 of his work he says: "The symptoms of uræmic intoxication are neither due to urea nor to any other constituent of the urine, nor can they be produced by the entire series of excretory materials contained in this fluid in their existing condition; they arise solely from this, namely, that the urea in the blood within the general circulatory system, under the influence of some special ferment, is decomposed into carbonate of ammonia. This carbonate of ammonia is the pernicious principle which produces these functional disorders; its injection into the blood will evoke all those symptoms which we recognize under the term uræmic."

---

[1] Guy's Hospital Reports, 1836, p. 360.

[2] On the Nature and Treatment of Diseases of the Kidney connected with Albuminous Urine. London, 1850.

[3] Die Brightische Krankheit und deren Behandlung. 1851.

Frerichs further adduces clinical proofs of the innocuousness of the accumulation of even large quantities of urea in the blood, and endeavors to support his own theory by experiments.

"(1) It requires to be proved," he writes, "that in every case of uræmic intoxication a decomposition of urea into carbonate of ammonia takes place; and (2) that all those symptoms which are held to characterize uræmia can be produced by the artificial introduction of carbonate of ammonia into the blood."

The first part of the argument which he proposed to himself, Frerichs sought to prove in this way: he removed both kidneys from some dogs, and then injected two or three grammes (from 31 to 46.5 grains) of urea into their veins. Convulsions and coma followed on this. In some cases there were no convulsions, and, in their stead, sopor and coma supervened; but before this vomiting came on, and, at the same time, ammonia could be demonstrated in the animal's breath. After death (which happened in from two and a half to ten hours after the injection of the urea) large quantities of ammonia were in each instance found in the blood. Carbonate of ammonia was further discovered in the stomach contents, the gall, and the other secretions. Finally, in the air expired by persons suffering with kidney disease, and in their blood when dead, Frerichs demonstrated the presence of ammonia.

The second part of his task Frerichs endeavored to accomplish by injecting a solution of carbonate of ammonia into the veins of animals. As a result of this be observed convulsions, which soon gave way to stupor, and he also discovered ammonia in the expired air.

At first this theory of Frerichs was accepted by general consent, although Henle[1] objected that it was merely a new riddle propounded instead of the old one, for in it Frerichs required a special ferment in the blood of the living individual, equal to the task of decomposing urea, but yet did not demonstrate the nature of this ferment, or the manner of its development.

---

[1] Handbuch der rationellen Pathologie. 1851. Bd. 2, Abtheil. 2, S. 321.

This was the deficiency which Treitz[1] sought to repair when he submitted the following proposition: the urea accumulated in the blood, when its excretion is obstructed through the normal channels, pervades the entire body and passes into all its secretions; but it appears most generally and in largest quantity on the mucous surface of the intestines. This urea, however, is regularly decomposed into carbonate of ammonia by the intestinal fluids, and this salt again is reabsorbed and carried back into the blood. Thus Treitz explains how "ammoniæmia" is brought about, and he adds that the same thing can be effected in a more direct way by the absorption of decomposed and ammoniacal urine from the urinary channels.

Jaksch,[2] in the following year, somewhat weakened the strength of the published views of his colleague, Treitz. He distinguished between uræmia and ammoniæmia as strongly as he could, upon clinical grounds, but still allowed the possibility of an ammoniæmia in the sense intended by Treitz in his theory. In the mean time objections were raised from various quarters against the doctrine propounded by Frerichs. The instances became constantly more numerous in which, notwithstanding the most careful analyses, no ammonia could be discovered either in the inspired breath or in the blood of uræmic persons. It was as early as 1852 that I myself first observed a case, afterwards published by Dr. von Kaup and Juergensen[3] in which, in spite of uræmic coma and repeated epileptic convulsions in a subject of genuine atrophic kidney, whose face even was covered with crystals of urea, no trace of ammonia could be discovered in either the breath or the blood drawn while he was alive. It began to look as if we should be obliged to reject the idea that uræmic convulsions depended upon the presence of carbonate of ammonia in the blood, or even that the introduction of this salt into the blood must of necessity produce, in the animals subjected to this experiment symptoms resembling those of uræmia.

---

[1] Ueber urämische Darmaffection. Prager Vierteljahrschrift. 1859. Bd. 4, S. 143.

[2] Klinische Mittheilungen. Prager Vierteljahrschrift. 1860. Band 2, S. 143.

[3] Deutsches Archiv für klin. Med. Band 6.

These contradictory statements and the doubts to which they gave rise led to a fresh series of inquiries being undertaken with the hope of solving the problem, but with results again that were not entirely reconcilable one with another. While some, who experimented by injecting carbonate of ammonia into the veins of animals, produced that full train of symptoms held to be characteristic of uræmia, others were only able by the same means to excite symptoms of irritation, such as restlessness, vertigo, vomiting, rigors, and convulsions, but failed of obtaining that most significant symptom—coma.

Schottin has already, in an early work of his upon the excretion of urea in the sweat,[1] raised some doubts upon the assumed conversion of urea into carbonate of ammonia within the blood-vessels; and in his paper on the symptoms which should characterize uræmia,[2] he showed that the sulphates of potash or of soda injected into the veins of an animal would produce exactly identical nerve symptoms with those that followed the injection of ammonia carbonate. To lessen the importance of the fact established by Frerichs—namely, the presence of carbonate of ammonia in the breath of uræmic patients—he called attention to the fact that this substance can be found in the breath of any person in whose mouth the natural secretions collect and pass into decomposition—as, for example, in typhoid states, pyæmia, etc. Schottin then tested repeatedly the breath of fifteen patients in whom all the so-called symptoms of uræmia were established in the clearest manner, but was unable to discover any carbonate of ammonia in it, although in the breath of another uræmic person, who had been lying for some time in a drowsy state and whose lips and gums were encrusted with dried sordes, he also was able to find a small quantity of ammonia. Schottin is of opinion that, in Bright's disease, urea can appear in the saliva of the mouth and can occur in the serum that transudes into the pulmonary tissue and produces œdema, and that this urea can then decompose, and so, from one or the other of these two sources, furnish ammonia to the breath. But when the presence of this substance is substantiated in the breath, it in no wise justifies the assumption of a decomposition of urea within the blood-vessels. The vomiting, too, of ammoniacal fluids is not admitted by him as proof of a previous formation of ammonia in the blood, but rather as evidence that urea, which has found its way into the interior of the stomach, has there become decomposed; and he supports his argument by the circumstance that unaltered urea is excreted by uræmic persons with the saliva, and with the serum which is poured out into the serous cavities, while ammonia is not found in these fluids.

---

[1] Archiv für physiologische Heilkunde. 1851. Bd. X. S. 469.

[2] Ibidem. 1853. Bd. XII. S. 170.

Schottin arrives at the conclusion that the urea, accumulated in the blood and combined with it, cannot of itself excite uræmic symptoms; but he brings into conspicuous relief the fact that other important substances, as well as urea, are excreted in some quantity by the kidneys. In one case, where a person died of fattily degenerated kidneys, he found the quantity of albumen to extractive substances in the blood serum standing in the following proportion—100 : 40; whereas in normal blood serum the ratio should be as 100 : 5, and he attributed the cause of uræmic symptoms in renal disease to an arrest of the tissue changes (Stoffmetamorphose), to a disturbance of the endosmotic and exosmotic processes which should take place between the blood and the tissues, and perhaps, also, to a general diminution of the oxidizing powers of the blood. As one of the reasons for this diminished oxidizing faculty in the blood, he reckons the diminution of its alkalinity, since it is sufficiently established that the combination of certain elements with oxygen is exceedingly promoted and quickened by the presence of alkalies.

Oppler,[1] who worked under Hoppe-Seyler's direction, arrived at similar results. In the first place he maintains that the symptoms produced by the injection of carbonate of ammonia into the veins of animals should not be regarded as identical with those observed in uræmia. Carbonate of ammonia in the blood produces symptoms of irritation in the nervous system, but not those signs of depression which are so characteristic of uræmia, namely, coma. Oppler was unable to detect the faintest trace of carbonate of ammonia in the blood of animals which he had rendered uræmic, either by extirpation of the kidneys or by ligature of their ureters. He found ammonia only once in the blood derived from the body of an animal after its death; but in regard to this he calls attention to the fact, long since ascertained, that after death ammonia is formed very quickly in the blood. Oppler, too, found an enormous increase of extractives in the blood of his animals, and a large quantity of kreatin (2.2 grammes in two pounds) as well as leucin in their muscles. He gave as the reasons for this increase of the extractives in the blood, not merely their retention in the system, in consequence of the suspended functions of the kidneys, but also the process of decomposition which is set in action in various organs on account of the altered composition of the blood.

---

[1] Beiträge zur Lehre von der Urämie. Virchow's Archiv. Bd. 21, S. 260.

Hoppe-Seyler before him had arrived at like results, finding, as he did,

1.27 parts urea,
8.60 " other extractives, } in 1,000 parts of blood serum

taken from the blood of a uræmic subject (after cholera) who had been bled. In the muscles of the same patient, after death, he found 1.59 grammes of kreatin to each pound by weight. From this Oppler considers it proved to demonstration that, as a result of the abrogation of the renal functions, enormous quantities of decomposition-products arise and accumulate in the muscles, and he considers himself justified in concluding that in the nervous centres, which stand affected by the self-same noxious elements, similar alterations of chemical composition must be brought about, although what these may be is at present unknown to us, from our ignorance of the nature of the decomposition-products of nerve waste. The soundness of every organ and its ability to fulfil its functions are, however, associated with the normal performance, throughout its substance, of the processes of endosmosis and exosmosis, and no organ resents any disturbance of its nutrition more certainly or more distinctly than does the brain.

Oppler considers it a decided error, that in attempting an elucidation of the uræmic symptoms, we should start with the idea that some one or other urinary constituent, or the product of its decomposition, acts as a deleterious substance in the blood, and ought therefore to be held guilty of the uræmic complex. He would seek the cause of these symptoms rather in some change in the chemical composition of the central organs; and with the results of his researches those more recently made by Perls,[1] of Königsberg, agree, as well in substance as in the conclusions which they lead to—conclusions which the last-named author found himself forced to adopt.

Petroff,[2] from Kasan, arrived, however, at totally different results. His experiments on animals convinced him:

1. That the injection of carbonate of ammonia into the blood produces symptoms exactly like those of uræmia.
2. That the gravity of the symptoms and their exact characters depend upon the quantity of ammonia in the blood, and upon the form or state in which it is present in that fluid.
3. That carbonate of ammonia does form in the blood when the kidney functions are abrogated.

Petroff experimented in the following way: he extirpated the kidneys of some animals, and in this state either left them to their fate, or further injected urea, carbonate of soda, or carbonate of ammonia into their blood; while, in other animals, without depriving them of their kidneys, he introduced solutions of ammonia salts in various doses into their systems. Petroff explained the differences in the results (as regards the demonstration of ammonia in the blood of animals which had been simply nephrotomized), arrived at by Oppler and himself, upon the ground of their different methods of experimenting. The fact that the simple injection of ammonia without prior nephrotomy should have been followed by no symptoms quite like those of uræmia, he explained by the circumstance that nephrotomized animals excrete carbonate of ammonia far too rapidly for it to produce any other than its primary symptoms of irritation; it has not time to evoke that depression of the nervous system which is itself so characteristic of the uræmic state. In this connection it is worth mentioning that Petroff, in the blood of three of his nephrotomized animals, was only able by his method to discover on an average 3 parts of free and 1.7 part of combined ammonia in each 10,000 parts of the blood, and this although two of the three creatures exhibited well-marked uræmic symptoms.

---

[1] Beiträge zur Lehre von der Urämie. Königsberger medicinische Jahrbücher. 1864. Bd. 4, S. 56.

[2] Zur Lehre von der Urämie. Virchow's Archiv. Bd. 25, S. 91

Zalesky, who made his experiments in the same manner, discovered even a less quantity, and attributed no importance to it whatever in the production of uræmic symptoms.

Kühne and Strauch[1]—who experimented on animals with the object of deciding this question, rendering them uræmic, partly by extirpation of the kidneys, and partly by ligature of the ureters—were unable to discover a trace of carbonate of ammonia in the blood of the dying creatures, and pronounced themselves thus positively: "As to whether or not the blood in uræmia contains carbonate of ammonia, we are able to answer, without hesitation, no."

On the other hand, Spiegelberg,[2] by means of Kühne and Strauch's own method, discovered a notable quantity of carbonate of ammonia in the blood of a pregnant woman attacked with eclamptic convulsions before the commencement of parturition. This blood, however, contained at the same time 0.055 per cent. of urea; and the urine of the same patient, drawn off by catheter just before the venesection (instituted for the purpose of obtaining blood which could be submitted to analysis), contained 4.78 per cent. of albumen and only 1.1 per cent. of urea. This observation encouraged Spiegelberg to institute, in common with Heidenhain, an entirely new series of experiments with the injection of carbonate of ammonia into the veins of animals; and the almost constant result of these carefully varied experiments was the production of symptoms exactly like those of eclampsia, namely, convulsions, with profound and persistent subsequent depression.

---

[1] Centralblatt für die medicinischen Wissenschaften. 1864. Nos. 36 and 37.

[2] Ein Beitrag zur Lehre von der Eklampsie. Bd. 1, S. 383. Berlin, 1870.

Rosenstein, who was not satisfied with the mere qualitative presence of carbonate of ammonia, as put forward by Spiegelberg in his case of eclampsia, and was unwilling to accept it in proof that the symptoms actually depended upon ammoniacal blood poisoning, himself made a further series of experiments on animals by injecting carbonate of ammonia. The results of his investigations, which in many respects were of the highest possible interest, were published by him, together with two cases of uræmia which had come under his observation in his Clinic, and in which no ammonia was found in the blood, although most carefully searched for. His observations led him to conclude that carbonate of ammonia, introduced in sufficient quantity into the blood, is capable of producing a complex of symptoms corresponding fully with that which characterizes epilepsy, and consequently with that group of symptoms which we observe in a certain number of cases of uræmia.

The convulsions excited by ammonia are doubtless of cerebral origin—indeed, are likely enough to be the direct effect of this poison upon the nerve substance of that portion of the brain which is the centre of convulsive actions; and of this much we may be certain, that these spasms are not produced reflectively through impressions conveyed either by the sympathetic in the neck or by the pneumogastrics. A condition of antecedent narcosis, compelled by morphia, chloroform, or chloral hydrate, exerts no influence whatever in preventing the convulsions; whereas narcotics, at least in the instance of puerperal eclampsia, appear to have favorable (antagonistic) effects. The poison is eliminated by the lungs only in insignificant quantity.

Indeed, the principal difference between the action of carbonate of ammonia and that of the agent which provokes uræmia rests in this, that the first mentioned is only empowered to evoke that complex of symptoms constituting epilepsy, whereas the latter produces both the symptoms of epilepsy and those of coma, convulsions and delirium. Still, even in those very cases of uræmia in which the character of the malady resembles exactly that which can be produced by ammonia poisoning, furnishing, as it does, epileptic features, and although, in a certain few of these cases, carbonate of ammonia may be found in the blood, the reference of the epilepsy to this ammonia cannot be entertained, since exactly the same symptoms may occur in man without any ammonia being found in his blood, as we learn from the investigations of Rosenstein and others, whose observations were conducted with scrupulous care.

While these efforts were being made to show the dependence of uræmia on some contamination of the blood either with urine elements or with their derivatives, and to refer its outbreak to some chemical influence at work upon the nervous system, investigations were instituted in an entirely different direction. In these efforts, in which the influence of chemical agencies in bringing about that array of symptoms called uræmia was simply not admitted, the aim was to seek and find out the essential cause of uræmia in local lesions of the central organs of the nervous system.

I previously considered the views of Osborne, who held that meningitis was the cause of uræmia, because he had found the pia mater somewhat opaque. Owen Rees started with the supposition that the blood of uræmic persons is always more watery than that of healthy individuals, and that to this fact is to be attributed the dropsical effusions in patients suffering from renal disease. When the great nerve centres become implicated in dropsy, we have, he says, quite enough to explain the uræmic symptoms. Unfortunately he adduces no grounds for the opinion here entertained.

This is the theory, however, although in somewhat modified shape, which Traube and his school took up again and further expanded. Traube[1] explains the occurrence of the uræmic attack as follows: before it comes on there has always existed a predisposition to serous effusions, arising from the thinning of the blood serum, which is so customary in kidney disease. Soon after the renal affection is established, an hypertrophy of the left ventricle of the heart fills up the sum of the offence, by dint of which an abnormal excess of pressure throughout the arterial system is effected at the same time as the blood serum itself is impoverished. If now, by any accidental circumstance, the blood tension becomes suddenly increased, or the blood serum is still further thinned, œdema of the brain ensues. But since the escape of the aqueous part of the blood takes place under the prevailing mean of pressure maintained in the aortic system, a pressure which is above that which obtains in the capillary vessels and veins, these last must suffer compression, and their actual blood contents be reduced in volume, to correspond with the amount of watery fluid effused; and the necessary consequence of this is anæmia of the brain.

---

[1] **Gesammelte Beiträge zur Pathologie und Physiologie. 1871. Bd. 2, S. 551.**

"The form of the uræmic attack will therefore depend on this, whether the whole brain or only certain portions of it are implicated in the process above described. If the cerebral hemispheres alone are rendered œdematous and anæmic, the patient becomes simply comatose, whereas if the middle brain [thalami and corpora striata] is similarly affected together with the hemispheres, convulsions as well as coma will appear. Finally, convulsions will be observed without coma, as in the well-known case reported by Bright, when the anæmia is restricted to the middle brain."

To strengthen his hypothesis, Traube alleges—

I. That he never saw an attack of uræmia in renal disease, where the left ventricle was not hypertrophied, and where an increase of tension in the aortic system could not be demonstrated.

II. That in the cases which he had seen, the diluted state of the blood serum could be recognized, even before the advent of uræmic paroxysms, either by the presence of dropsical effusions, or, in the absence of these, by the pallor of the skin and those portions of the mucous membranes which are within sight.

III. That in every instance in which he had carefully examined the contents of the skull cavity after death, he had been able to confirm the existence of more or less considerable œdematous swelling of the brain as well as distinctly marked bloodlessness.

IV. That, furthermore, the presence of blood effusions within the cranial cavity, observed in many of these cases, confirms the suspicion that the abnormally high arterial blood pressure to which these effusions owe their origin has also something to do with the production of the œdema, which is present at the same time.

V. That it should be remembered that the well-known experiments of Kussmaul and Tenner have shown that convulsions and coma can be produced in animals by cutting off the supply of arterial blood to their brains.

Ph. Munck[1] endeavored to support Traube's theory by experiments. He first tied both the ureters of a dog, then ligated the jugular on one side, and shortly afterwards injected water into the carotid upon the same side. Upon the first essay of this experiment, the animal almost immediately fell into a comatose state, with some twitchings of muscles, and afterwards exhibited the most violent convulsions. Every repetition of the experiment was attended by very nearly the same results, sometimes convulsions and at others coma predominating, just as happens in uræmia. After extirpation of the kidneys, too, the symptoms followed the injection of the water almost as rapidly as after ligature of the ureters. Any great loss of blood or violent vomiting, in the first few hours after the water had been injected, caused a marked remission of the symptoms. The injection of fresh defibrinated blood was followed by the same results. The pathological appearances found after death were œdema of the brain without extravasation of blood.

When Munck ligated the ureters of animals and afterwards tied their carotids, they evinced no sign of either coma or convulsions up to the moment of their deaths, although they had vomiting, and, as death was approaching, the greatest irregularity of pulse and respiration. After death, the anterior and middle parts of the brain were found anæmic, the posterior part, and especially the medulla oblongata, showing a high degree of arterial congestion.

The conclusion drawn was, that thinning of the blood, occurring at the same time with increased arterial tension, excites that complex of symptoms which we call uræmic.

In animals, coma and convulsions usually do not follow the diminution of the blood supply to the middle brain and hemispheres, after the ureters have been ligated, whereas the symptoms, which are excitable through the cerebellum and medulla oblongata, then manifest themselves with remarkable distinctness.

---

[1] Ueber Urämie. Berliner klin. Wochenschrift. 1864. S. 111.

Dr. W. Rommelaere, fellow of the Royal Society of Medicine and Natural Philosophy in Brussels, has subjected to a critical examination all the theories which have been proposed regarding the essential causes of the uræmic symptoms, and his inquiry is all the more valuable, for the reason that he has not shirked the trouble of repeating every one of the experiments which other men had made before him, with the view of settling the question and of testing the correctness of the results which they have published. At the conclusion of his work[1] he makes the following statement:

"All the theories which seek to explain the occurrence of uræmia, through a retention of some one or other constituent of the urine in the system, are not in harmony with the facts.

"The injection of various substances into the blood-vessels may lead perhaps to some discoveries in pathogenesis, but it would be a blunder of the first magnitude for the physician to establish his theory of uræmia upon the results of these experiments; this must be based on facts furnished by clinical experience alone."

Rommelaere especially points out, by clinical observations, that the assertion that the blood of uræmic persons always contains carbonate of ammonia, is an erroneous one. In point of fact, but few observers have succeeded in discovering carbonate of ammonia in the blood. The single conclusion which we can accept from the observations we at present have is, that there are cases of uræmia in which ammonia is contained in the blood, and others, of apparently the same nature, in which chemical analysis has hitherto entirely failed to detect a trace of that substance.

Then too, from the results of the experiments made by him on animals, by tying the four jugular veins and injecting water into the blood, Rommelaere is unable to express himself in a manner favorable to the theory of Traube.

---

[1] De la pathogenie des symptômes urémiques. Étude de physiologie pathologique. Journal de Médecine, etc. Vols. 44 and 45. Bruxelles, 1867.

Rommelaere proposes the following formula to help us in elucidating the etiology of uræmic symptoms: the suppression of the urinary secretion prevents the further elaboration of albuminoid substances throughout the entire organism, if nature does not provide some other vicarious mode of supplementing this excretion. Urea is the last step or stage of the metamorphosis of these albuminoid substances, and, when urea accumulates in the blood, it changes the character of this fluid, and consequently the vital (nutritional) processes in various parts of the body. The entire work of transforming the albuminoid substances is arrested, so that we have to deal with the retention in the system, not merely of urea but also of nitrogenous substances in all the different stages of oxidation through which they pass within the organism.

As one of the principal results, albeit only a negative one, arrived at by the experimental physiologists in their endeavors to solve the question, Rommelaere mentions the fact that we can no longer accuse any one substance that may occur in the blood, after suppression of the urinary excretion, of being alone and by itself the cause of the nervous symptoms. The analyses of the blood, undertaken in states of uræmia, have furnished dissimilar results: at one time ammonia is found, at another it is absent; at one time the blood is overloaded with urea, at another it is not; and the pathological changes found in the brain after death from uræmia vary to an equal degree.

One thing alone we may accept as invariable, upon Rommelaere's authority, namely, that the maladies which entail uræmic disturbances, are characterized, at the time when the uræmic complications break out, by the presence in the tissues of the body of an excessive amount of nitrogenous excretory materials. At the same time Rommelaere cannot, if we may judge from the results of his experiments, dispute the fact that a preponderance of some one of these excretory elements, over and above the rest, may, at a particular time, exert a greater or less influence in producing the uræmic attack. Thus he holds it to be quite possible that the presence of ammonia in the blood may give rise to cramps, or that hydræmia, in combination with increased arterial pressure, may determine coma. Convulsions and coma, however, may arise without the presence of ammonia in the blood, and without hydræmia, acting in conjunction with increased blood pressure in the aortic system.

We must look for the cause of uræmia in an abatement of the activity of the kidney functions in their entirety, or—in other words—in the failure of these organs to excrete, not merely the urinary water, but also the nitrogenous waste of the tissues.

Lastly, Voit has appended to his lengthy work[1] on the behavior of kreatin, kreatinin and urea in animals, some remarks upon uræmia. He describes as uræmic those symptoms of disease which supervene when the excretion of those waste materials (resulting from interstitial changes) which cannot be converted into gaseous products is either suppressed or is insufficiently accomplished. He shows that the urea in the body is not nearly so quickly decomposed into carbonate of ammonia as has been ordinarily supposed. There is only one part of the body where rapid decomposition of urea into carbonate of ammonia takes place, and that is the mucous membrane of the whole of the intestines. Armed by his own observations, Voit rejects Frerichs' explanation of uræmia, as a theory to be adopted to the exclusion of every other, or as one adapted to meet every case, quite as decisively as Rommelaere does.

Voit's experiments appoint with certainty that urea by itself may well be a perfectly harmless substance, and may pass into the body without inducing any symptoms whatever, and yet lead to the most violent disturbances when its elimination is prevented for any length of time. He and Oertel, in experimenting on a dog, which they had fed with 18.4 grammes of urea added to his meat, giving him the same diet daily, with abundance of water, found that the animal eliminated the whole of it readily without experiencing the slightest disturbance ; but as soon as they withheld water from the beast entirely, while still adding to his food the same doses of urea which he had previously perfectly well supported, all the symptoms of uræmia appeared—vomiting, convulsions, sopor, and profound prostration of the system generally. All these symptoms disappeared, as they do in the so-called typhoid state of cholera, directly the urea was once more excreted from the system. It is worth noticing, however, that, while the uræmic symptoms endured, there was no carbonate of ammonia discoverable in the animal's breath, although the last portions of food rejected by vomiting gave a strongly alkaline reaction and contained ammonia.

---

[1] Zeitschrift für Biologie. Bd. 4, S. 140. München, 1868.

According to Voit's opinion, quite a considerable quantity of urea can be held in the blood without occasioning uræmic symptoms; but then arises the question, what is this quantity?

"Symptoms of disease originate whenever any substance, which does not belong to the composition of the economy, accumulates in any quantity within the body, and is not eliminated from it. In suppression of the urinary excretion it is not one single element, like urea or uric acid, kreatin or kreatinin, the extractives, or urochrom [pigmentary matter], which does the harm; it is the mass together. Under similar circumstances some extraneous salt, like carbonate of ammonia or Glauber's salt, etc., would produce the same symptoms."[1]

Herein the views of Voit agree entirely with those expressed by Hoppe-Seyler, and also by Buhl, upon the origin of the symptoms in the typhoid stage of cholera.

Voit does not dispute the possibility of a watery condition of the brain being able to provoke the so-called uræmic symptoms; but he maintains that death, as it ordinarily happens when urinary substances are retained in the system, is not provoked by any such brain œdema.

Voit considers the term uræmia an incorrect one. The question is not simply one of mere accumulation of urinary constituents in the blood. "The smallest component particles of organs [the ultimate tissues], which are constantly exchanging materials with the nutritive fluids, are the seats of re- and decompositions and vital phenomena, and when the products of metamorphosis, thence derived, are not swept away, but accumulate *in situ*, these very capillary interchanges, of which the osmotic currents constitute the principal factors, are no longer able to take place properly, and a stagnation of them ensues."

---

[1] Voit dosed a large dog with a considerable quantity of benzoate of soda added to its food, and observed just the same symptoms as are produced by large doses of urea or by retention of urine.

In regard to the potent and striking effect of potash salts upon the system, when they are introduced into the blood, Voit expresses his conviction that it is the excess of potash, derived from the retrograde metamorphosis of muscle (Fleisch) in the body and its arrival and retention in the blood plasma, which is in a large measure concerned in provoking the symptoms of uræmia.

The strenuous efforts made by such a host of inquirers to discover *the essential nature of uræmia*, and the conflicting views to which the labors of the different experimenters have led, afford the best proof of the difficulties which stand in the way of a solution of the problem. One of the chief reasons why we are compelled, even at the present day, to confess our inability to solve this question, lies, as I believe, in the circumstance that our attention has been chiefly devoted to experiments upon animals—a useful enough adjunct in our researches—while the prime source of knowledge, clinical observation, has been all too little tapped.

Guided by one-sided, preconceived ideas, the larger half of the experimenters have, without further hesitation, identified the results of most gross interference with the organisms of animals, with the events which follow in the human body when the functions of those most important secreting glands, the kidneys, are persistently abrogated. They considered themselves justified in drawing such a conclusion, because there was some external likeness between the symptoms which are capable of being thus provoked in animals and those produced in man by the sudden or gradual arrest of the renal secretion. Yet, surely, one has as much right to assume that a series of epileptic seizures, following rapidly one upon the other in an old epileptic subject and succeeded by coma, are uræmic manifestations, as to identify the after effects of an extirpation of both kidneys, or a ligature of the ureters, with a simple renal affection uncomplicated by any grave wounding. Further, I am not disposed to attach much value to the sudden introduction of a solution of this or that foreign substance into the blood circulating in the vessels of a healthy animal, or to the sudden elevation of the blood pressure in the vessels (up to a degree which does not admit of being calculated) by the injection of large quantities of water into the circulation—the natural sluices for the excretion of the foreign matters as well as of the water having been at the same time barred. Of a truth, before we shall be able to return any decisive response in regard to the etiology of uræmia, we must possess further and more minute clinical observations, fuller and more complete analyses of the blood of uræmic patients, more accurate investigations into the chemical composition of the interstitial serous fluids, especially of that found in the brain; and these, perhaps, may prove that symptoms, which we have accepted as the results of one and the same cause, have been brought about by a variety of different ones.

Now, as regards my own experience at the bed-side, I must admit that the first example of uræmia, in its full perfection, which I had the chance to observe in the year 1852, as the final dénouement of a chronic renal malady, made a strong impression on my mind in regard to one particular—the face of the patient became covered with crystals of urea just before the outbreak of the final, general convulsions, so that his beard looked frosted. Indeed the look of this fellow will never be blotted out of my memory, and has sufficed to maintain the firm conviction within me, throughout all the fluctuations through which the doctrine of uræmia has passed during these years, that when the quantity of urea in the blood has reached a certain extreme excess, it is capable of evoking or producing that entire complex of symptoms which we call uræmic. In this very case, unfortunately, no exact estimation of the quantity of urea contained in the blood after death was made. The observation of similar cases since that time has strengthened this conviction. In one of these, for instance, the urea, shortly before the attack of epileptiform convulsions, showed itself as an excretion upon the skin of the face. In two other instances, in which urea was deposited upon the skin, but which occurred in persons who had no kidney disease, there were no convulsions, and in one of them only was death preceded by a protracted condition of coma.

I have only once succeeded, after a first uræmic seizure has taken place, in examining the blood drawn from a vein. The case was one of a young, robust, journeyman butcher, and his blood contained a notable quantity of nitrate of urea; but in this instance, too, which happened in 1853, lack of proper apparatus prevented our estimating the urea quantitatively. The autopsy showed advanced contraction of both kidneys.

By the side of these cases, however, I am now able to range a number of others (of more recent date) which came under my own observation, where the blood, drawn either directly after the outbreak of uræmic symptoms or taken from the body immediately after death, has been subjected to careful analysis, and its content of urea has been estimated. In one example the blood held so little urea that Dr. Jacobsen (now professor of chemistry at Rostock), who was kind enough to undertake the analysis for me, could not estimate its amount quantitatively; in another case Dr. Jacobsen found the quantity of urea in the blood only 0.01 per cent.; in a third case, 100 grms. of blood furnished 1.6 grms. of nitrate of urea, making a content, therefore, of nearly 0.8 per cent. of urea.

From these observations it follows, *that the overloading of the blood with urea is certainly not, in every instance, the cause of the uræmic symptoms.*

In quite a large number of cases I have tested the blood drawn during life, after uræmic symptoms have supervened, by the Kühne-Strauch method (passing a stream of hydrogen gas through it, and then, by means of Nessler's reagent, testing the gases which pass over for the presence of carbonate of ammonia), but have always failed to find carbonate of ammonia. My own observations, therefore, as well as those of other observers, and of Rosenstein in particular, show conclusively that *Frerichs' theory can only be the correct one for at most a few cases of uræmia.* In one case I was even able to establish the entire absence of carbonate of ammonia in the blood, when only just before the venesection was made the contents of the patient's stomach, rejected by vomiting, smelt of ammonia, and gave a strongly alkaline reaction.

Hitherto I have not examined the blood of uræmic patients for any of the other nitrogenous substances which normally are destined to be excreted. On the other hand, in a number of cases of uræmia I have endeavored to determine the proportion of water contained in the blood serum (in the earlier cases, it is true, this was done by simply testing the specific gravity of this fluid and inferring, from the low figure at which it stood, that the watery content was excessive).

The figures marking the specific gravity of the serum of the blood of uræmic patients, drawn by venesection or taken from their bodies after death, are shown in the following table for each of the cases :

		Solid Constituents.
St., uræmic convulsions, after one attack of . .	1016.8	——
" " " " two attacks of.	1018.1	——
H. H., " "	1022.8	8.84 p. ct.
Lassen," "	1021.	11.23 "
Kn., " "	1023.4	12.85 "
H., " "	1024.3	9.34 "

The specific gravity was in all these cases ascertained by means of the pyknometer.[1] Only the second specimen of blood examined, that from the patient St., was taken after death ; all the other estimates have reference to blood drawn during life by little venesections. In December, 1873, in examining the blood serum, obtained by a small bleeding from a patient five hours only before death, when he was in a comatose state with uræmic twitchings, I met with a far higher specific gravity than in the cases above tested ; the serum here marked 1030.58 sp. gr. (vide infra, Case VIII.).

Now does it follow, as the result of these inquiries, that the symptoms of uræmia exhibited by these patients were due to the watery condition of their blood-serum ? Certainly not ; for the very last case I instanced shows that uræmic symptoms can arise when the blood serum is of normal specific gravity ; and, again, I have taken blood from another patient suffering from renal disease, in whom, however, there were no symptoms of uræmia, and found the serum even more watery than I ever yet encountered it in uræmia, presenting a specific gravity of only

---

[1] Vide *Hoppe-Seyler's* Handbuch der physiologisch- und pathologisch-chemischen Analyse. Berlin, 1865, pp. 15 and 16.

1015.58, and containing only 4.3 per cent. of solids (vide infra, Case XVI.). In the patient St., who furnished the blood for the first two tests enumerated in the list given above, where the specific gravity was so excessively low, the first series of uræmic attacks lasted for three days. Then, between the 7th of June and the 22d of August, the brain functions were normally fulfilled, and it was only after this interval of ten weeks that the second fatal attack of uræmic symptoms overtook him; yet the persistence of the dropsy through all this interval would not justify us in supposing that in the meantime the blood serum had become more concentrated.

Certainly the statement is correct enough that the left ventricle was found hypertrophied, and that there was a well-marked condition of contracted kidney in the bodies of all the persons whose blood had thus been tested, and all of whom, with one exception (St., who resisted two attacks), succumbed at once to uræmia. But one is not to conclude from this that excess of arterial tension, as the result of cardiac hypertrophy, and a watery state of the blood, are the indispensable conditions for the production of the uræmic state. In St., whose kidneys were in a condition of chronic parenchymatous nephritis, this hypertrophy of the left ventricle was absent.

Both the conditions laid down by Traube as requisite for the production of uræmia, namely, a watery blood and an elevated arterial blood pressure, are absent as a matter of certainty in the uræmic attacks following cholera, as has been demonstrated by Voit and others. Yet it is certain that these attacks fairly merit the title of *uræmic;* they come on after long-continued complete suppression of urine, and are not so rarely accompanied by excretion of urea upon the surface of the skin.

I have not omitted to pay due heed to the state of the brain in the bodies of my uræmic subjects. But in this investigation I at once encountered great difficulty in determining the degree of serous infiltration of the brain substance. So far as I am aware, mere naked eye observation or the sense of touch will not suffice, in most cases, to determine whether a brain be or be not richer in watery elements than it should be. Nothing but direct estimation of its watery contents, by ordinary analysis, could give us positive information upon the fact; and this has never, to my knowledge, in the state in question, been resorted to. Unfortunately, I too have hitherto neglected to make the requisite analysis. Cases, however, of death have come before me where the uræmic symptoms were exquisitely complete (epileptiform convulsions, followed by coma), yet at the autopsy no abnormal saturation, with serum, of any part of the brain could be discerned, estimating this after the ordinary way by looking at it. Cohnheim, who was my colleague at that time, made a post-mortem examination of such a case on July 11, 1871; and since it is in many respects interesting, I will briefly narrate it here.

CASE VI.—Caroline Lassen, housemaid from Kiel, twenty-two years of age, was admitted into the Medical Clinic, July 5th, 1871. She had kept at her work nearly up to the date of her admission, but had been ailing for some time. Some days before her admission she had been compelled to take to her bed, on account of severe vomiting of blood. The patient could tell us no more than this.

Condition on admission.—Remarkable pallor of face and skin generally. Moderate anasarca of face, trunk, and upper extremities; considerable œdema of lower limbs. No ascites. Patient's memory is bad; she is morose and very sparing of her words; complains only of headache and a sensation of pressure in the region of the stomach. The breath is excessively fetid; heart dullness increased; loud pericardial friction sound; no lesions found in the lungs; urine pale, rather hazy, faintly alkaline, specific gravity 1007, highly albuminous.

During the next few days the patient vomited repeatedly, although she had taken nothing but a little fluid. The vomit, with which small quantities of blood were mixed, presented an alkaline reaction, but did not smell of ammonia. The general œdema began to subside, and the patient appeared less apathetic; but, soon after, her mind again became somewhat clouded.

On the 9th of July, constant vomiting. Patient's breath and the substances vomited spread a most penetrating and abominable odor, although in neither was there any ammonia to be discovered. She complains of headache; drinks plenty of water but eats nothing.

On the morning of the 10th inst., at 7 o'clock, she had a violent epileptic attack. She was bled to the amount of 150 grms.; afterwards recovered herself sufficiently to help herself to a considerable quantity of fluid. At 12 o'clock she had a second seizure, when she was bled again; death followed speedily.

During the six days she was under observation this patient passed altogether 4,900 c. ctm. of urine, a daily average therefore of 816.66. The specific gravity of the first five days' quantity was 1010, that of the sixth day 1011. Its reaction was always alkaline. The sediment contained only very few, and these perfectly hyaline casts. The content of urea and albumen was only reckoned for three days. The urine quantity for these days, reckoning that of the sixth day in, when the specific gravity was 1011, amounted altogether to 2,330 c. ctm., and it contained 24.275 grms. of urea and 7.259 grms. of albumen. The specific gravity of the urine remained so constant on each day it was examined, that one may, without hesitation, reckon a similar amount of urea to have been passed on the days when it was not specially analyzed. This would make a total excretion of 51.05 grms. of urea for six days, or for each day respectively 8.51 grms.

The venous blood drawn before death separated into a loose coagulum and a milky, cloudy serum, whose specific gravity, taken by the pyknometer, was 1021, its water contents being estimated to be 88.77 per cent. From 100 c. ctm. of the blood (clot and serum together) an alcoholic extract was made; this was dried; the sediment extracted a second time with alcohol, dried again; the residue treated with water, with addition of concentrated nitric acid; from this 1.6 grm. of nitrate of urea was obtained, which makes the blood to hold very nearly 0.8 per cent. of urea entirely apart from the unavoidable loss which the process must have entailed. Of the smaller portion of blood obtained at the second bleeding, 50 c. ctm. were taken, and treated exactly in the same way. Only the filtrate derived from the last watery extract, after previous decomposition with baryta solution, was tested by Liebig's solution of nitrate of mercury, in order to estimate the urea; and in this way it gave as result 1.42 per cent. of urea.

The following account of what was found at the autopsy I take down in Cohnheim's own words: Slight œdema of both lower extremities; a long, oval, thin, and light skull-cap; sutures well defined; longitudinal sinus nearly empty; dura mater of normal thickness and natural polish; fluid blood only in the sinuses at the base; pia mater over the convexity of brain throughout slightly opalescent, but quite delicate and pale. Several lentil-sized, flat, and superficial extravasations into the tissue of the pia mater, not implicating the brain substance itself, were scattered over the upper portions of both hemispheres and at their sides, just where they begin to curve under towards the base. Ventricles of normal size; ependyma delicate; brain substance throughout of good consistence; on section it presented the gloss of moderate moisture. Both gray and white substances remarkably anæmic. No deposits or patches of disease anywhere. The same anæmia in both the cerebrum and the cerebellum, in the central ganglia, the pons, and the medulla oblongata. The blood discovered in the larger vessels of the pia mater remarkably watery and pale red. The pericardium contained three ounces of a whey-like fluid mixed with shreds of fibrine. Both pericardial layers, the external and the visceral, were coated with a soft, quite transparent colorless villous layer of fibrinous exudation. Heart, in its long diameter, somewhat larger than normal; left ventricle felt very hard; in both ventricles there was a small quantity of very loose fibrine clot; valves of both sides smooth; color of muscular substance pale; thickness of right ventricular wall 0.4, of left 1.6 ctm.; left ventricle moderately dilated, its musculi papillares thick and round; aorta narrow and of normal texture. Both kidneys considerably atrophied; the left, measuring in its long diameter, ten ctms., in its broad diameter four ctms.; the right eight ctms. in length and only three ctms. in breadth; both were also considerably thinner than normal. Capsules of both could be removed with some difficulty, and left an uneven surface upon being removed. This unevenness was found to be due to the presence of numberless fine and coarse granulations and nodules, separated everywhere by narrow furrows. The color of the elevated nodules was throughout white, that of the furrows between them pale red. On section, the kidneys were found to be pretty firm and quite extraordinarily pale, so that even the medullary cones themselves were only of a pale rose color; then, besides, the cortical substance was remarkably narrowed—that of the right kidney especially, where the bases of the cones nearly touched the kidney capsules, while the left presented a cortical stratum that measured only between one and two mm. in diameter. The mucous membrane of the pelvis of the kidney was pale. The stomach and duodenum contained some dirty brown fluid. The mucous membrane of the stomach was slightly œdematous, of a faint slate color, but perfectly smooth, without ulcer or scar. The mucous membrane of the rest of the digestive tract was smooth. The contents of the small intestines were pultaceous and yellow, and firm, brown, fæcal masses were found in the colon. In the more dependent parts of the abdominal cavity there were a few ounces of a perfectly transparent red serous transudation. I pass over the further details of the autopsy, since they are unimportant and of no further interest to us.

Now, in this case, was it the excess of urea, proved to exist in the blood, which produced the uræmic symptoms; or was it the excessive anæmia, observed both during life and after death? I am unable to decide between them; but one thing is certain, there was no œdema of the brain.

Considerable doubts as to the correctness of Traube's theory, or rather scruples against its applicability to every case of uræmia without reserve, have from the very beginning been excited in my own mind by the circumstance that uræmic attacks do not by any means occur chiefly in those cases of renal disease which are characterized by dropsy; indeed, so long as the dropsical swellings are still increasing, uræmic symptoms are, comparatively speaking, rarely observed. On the other hand, I have repeatedly noticed that uræmic attacks of the gravest kind have supervened in those very patients in whom I have accomplished a sudden absorption of dropsical effusions by means of some profuse water-drain, sweating or purging, and in whom the general anasarca has been thus rapidly subdued. The following is a remarkable example of this:

CASE VII.[1]—A robust, muscular countryman, aged thirty-six, who came from Ditmarsch, sought admission into the hospital here, on account of general dropsy, which appeared, by his account, to have lasted six or seven weeks. He had previously been laid up for eighteen months, off and on, with intermittent fever of a quartan type; but the fever had entirely left him before the dropsy began. The general surface of the patient's skin was of dirty gray, yellow color, and his mucous membranes, where visible, were pale. His digestion was not disturbed, and his appetite was good. By his own account, the urinary secretion had been scanty for some time past, and the very small quantity passed by him, in the first twenty-four hours after his admission, contained an enormous quantity of albumen as well as abundant hyaline casts and epithelial cells from the renal tubules; the specific gravity was 1033.

I was anxious in this case to try the effect of the sweat cure recommended by Liebermeister, in the hope of alleviating the extreme discomfort of his dropsy; for his scrotum was swollen to the size of a child's head, and the œdema of his foreskin had twisted up his penis into the shape of a post-horn.

To this end, on the day after he was admitted, the patient was placed in a hot bath, at a temperature of 39° Centigrade (102.5° Fahr.), and kept in it for rather more than half an hour, the temperature being kept up to the same point. He was then packed in woollen blankets, to keep up the sweating after the bath; and he broke into a most profuse perspiration all over. While the patient was lying streaming with sweat in this way, quite suddenly, without any premonitions, sharp twitching took place in the muscles of his face, which shortly resulted in a complete epileptiform attack. After the convulsions had ceased, and the fellow had lain for a short time in a comatose state, he opened his eyes, and passed into a state of acute mania: screamed and stormed, struck and bit his attendants, until a second epileptic attack ensued, to be followed again by a short interval of coma and another maniacal frenzy. In this way the symptoms repeated themselves four times within the hour; and then the patient recovered his senses, and, with the exception of some headache, of which he complained in the evening, as also of feeling dull and tired, he appeared quite well.

The following night he slept badly, but in the morning felt perfectly well. And now, to my no small astonishment, I discovered that the swelling of his scrotum and penis had completely subsided; while the skin over both thighs, which had but yesterday been tight and distended with anasarca, was loose and wrinkled. Now this patient had never experienced an epileptic attack before this, and, for the six weeks he was in the hospital afterwards, he had no further return of them. A further consequence of this first bath was a more abundant discharge of urine, whereby about thirty grms. of urea were excreted daily; subsequently I had no fear in carrying out the sweat cure according to the method above described, and this was borne by the patient without any unpleasant results;

---

[1] This case was previously published by me in the Greifswalder Med. Beiträge. Bd. 3, S. 58.

indeed, when he was discharged, at his own request, his urine, it is true, still contained albumen, but he was almost entirely free from dropsy.

The sudden outbreak of uræmic symptoms in this case I am only able to explain in the following way: the enormous loss of water which took place by the skin effected an absorption of the dropsical fluids contained in the subcutaneous cellular tissues and the serous cavities of the body. In consequence of this, the urea and excrementitious substances which were contained in these fluids, and held, as it were, in depots where they could produce no ill effects upon the general organism, passed back into the circulation, and, aided perhaps by the simultaneous action of the hot bath and elevated temperature of the body, produced the violent nervous symptoms already described—symptoms that subsided, it is true, upon the occurrence, immediately after the bath, of profuse urination, and which did not reappear so long as the flow of urine continued to be abundant—and this, too, notwitstanding the fact that on the very day following the attacks the hot bath was repeated. Surely no one would be disposed, in this case, to think of the occurrence of an acute and quite temporary œdema of the brain during the sweating process.

In our endeavors to test the correctness of Traube's theory by clinical experience, it is impossible to disregard the striking fact that uræmic symptoms often occur in persons suffering from renal disease, who never, either at the time of the attack or previously, have been affected with anasarca or dropsical accumulations in the serous cavities. If, notwithstanding this, Traube observed, with such regularity, œdema of the brain in the bodies of persons dying with uræmic symptoms, it must be remembered that another explanation of this can be given than that with which he has furnished us. Let us consider only the group of symptoms usually observed in a severe attack of epileptiform convulsions. Now such convulsions are always associated with grave disturbances of the circulation, which are rendered most distinct and apparent in the cyanotic coloration of the face, in consequence of the extreme blood accumulation in the vessels of the head. This hyperæmia, too, frequently leads to extravasations into the skin of the face, and into the conjunctival submucous tissue, and not rarely to the bursting of vessels in the brain substance itself, or in its membranes—as I have frequently seen in persons who have had epileptic seizures unconnected with disease of the kidneys. In the body of a child, only a year and a half old, who had been sick with whooping-cough, and had died after an attack of so-called eclampsia, I found an apoplectic clot, the size of a hazelnut, in the substance of the cerebrum. In the body of a primipara, twenty-three years of age, who had had eclamptic convulsions from the moment of parturition, and in the course of five hours had passed through sixteen complete epileptic attacks, I found several effusions of blood in the cortical parts of the brain, and over the convexities of both hemispheres, as well as bloody infiltration of the tissues of the venous plexus in the right ventricle, and bloody coloration of the serum contained in this ventricle. In the urine drawn off by catheter before the attack, I found that 1,000 c.ctm. contained 0.054 per cent. of albumen, and in a second portion, passed after the attack, the albumen content was (as much as) 0.234 per cent. The first portion had a specific gravity of 1013, and contained 1.04 per cent. of urea; the second was 1015, and contained 1.65 per cent. of urea; neither portion contained any casts. The kidneys, upon removal, were found to be perfectly normal. The albuminuria, probably induced, in the first instance, by the act of parturition, was, like the cerebral bleeding, produced by the disturbance of the circulation connected with the convulsions, and increased therefore with their prolongation.[1]

It appears to me that there is no escaping the conclusion, that the brain œdema, which Traube observed so invariably after uræmic symptoms, when these had culminated in convulsions, must have followed as a consequence of the spasms, and could not have been the cause of them. Why is it, one is forced to ask, that one misses all œdema, in other parts of the body, in so many instances of rapid death by uræmia? We are not aware that the brain-substance has any special proclivity, over all other tissues, to become œdematous. Then, too, in so many cases where the brain appears upon the post-mortem table soaked through and œdematous, and there are collections of dropsical serum in the ventricles and in the meshes of the pia mater, how comes it that no convulsions took place before death, and why is it that death, in these instances, is preceded by no further nervous symptoms than a gradually increasing state of stupor, such as any prolongation of the act of dying brings with it?

---

[1] Compare *Huppert*, Virch. Archiv. Bd 59, S. 367.

My own experience, gathered at the bedside, as well as the observations I have instituted, has brought me to the opinion that what we call uræmic symptoms and encounter in renal disease are not always brought about in the same way and do not admit of being explained by one and the same cause. *I only consider this much established, that the symptoms are all caused by some disorder of the urinary secretion, and that the title of uræmia is rightly attached to them.*

That uræmic symptoms do depend on some disturbance of the functions of the kidney, appears most plainly from their supervention in complete suppression of the urinary secretion, as may often be observed after scarlet fever and cholera, and, although more rarely, sometimes after diphtheria. Experience, however, teaches us that the excretion of urine may, in some instances, be interrupted for a considerable time, without anything of the kind occurring, in proof of which I may refer to Case III., previously detailed by me, in which there was absolute anuria of five days' duration. In cases where the commencement of uræmic symptoms is delayed, the secretion of urine being arrested, one must remember that the elements of the urine, both the water and the other excrementitious substances, admit of being removed by other channels besides the kidneys—for example, through the skin with the sweat,[1] and through the mucous membrane of the digestive tract by vomiting and diarrhœa (Bernard and Barreswill). But there are also cases in which, notwithstanding the persistence of absolute anuria for several days, vicarious excretions of the kind we have mentioned have not been fulfilled, and in which death occurs after quite a short agony, and without antecedent convulsions or true comatose symptoms. I have watched such a termination to a case of nephritis after diphtheria, and again in one instance of amyloid degeneration of the kidneys. It is true that in the last case complete suppression of urine had not taken place, but in the last week of life the patient passed altogether only 509 c. ctm. of urine, on some days not a drop, and had only excreted quite scanty quantities of mucus by vomiting.

---

[1] *W. Leube*, Ueber den Antagonismus zwischen Harn- und Schweisssecretion. Deutsches Archiv für klin. Medic. Bd. 7, S. 1.

Now from the extreme complication of the conditions we here encounter, and because of the obscurity in which the essential cause of uræmia still remains enshrouded, it has, up to the present time, been impossible to obtain more than an approximate idea of the degree of secretional suppression requisite for the determination of uræmic symptoms. Convinced, as I have been, that these symptoms can be produced through a retention of the specific constituents of the urine—the dross or waste derived from the decomposition of nitrogenous substances—in the system, I have directed my attention to ascertain whether any at all constant relation between the diminution of excretion of these substances and the advent of uræmic symptoms could be shown to exist. In these efforts I was obliged to restrict myself to the consideration of only one element, namely, the urea; the reasons for this being, first, that it stood foremost among the specific elements of the urine by its quantity, and then because by the amount of it present we estimate, upon well-established physiological principles, the waste of the nitrogenous substances taking place in the body in health; whereas we are totally ignorant of the ratio borne by the non-crystallizable urinary substances towards the varying energy of the capillary interchanges; and, lastly, because we possess a mode of quantitatively estimating this particular nitrogenous constituent of the urine (the urea), which presents no practical difficulties in its application.

I am well aware that the results of my work in this direction cannot claim to be of any great value. Even if the opinion which I hold be entertained, that a disproportion between the production and the elimination of urea from the human body is of itself enough to produce uræmic symptoms, we are still ignorant of the amount which would be required to do this.

In the first place, the production of urea will depend upon the quantity of nitrogenous substances present in the body and participating in the nutritional interchanges; in the second place, it will depend upon the quantity of nourishment taken and assimilated; and, in the third and last place, it will vary with a variety of conditions, which are, at all events, in a measure beyond our ken and powers of estimation.

These considerations show very clearly that a certain measure of urea, such as completely sufficed for a particular individual, and represented the total quantity of this substance formed in his body and eliminated from it daily, might yet fall below the measure which another person ought to produce.[1] The same discrepancy will hold good in comparing the observations made upon sick persons and those who are in health. In pursuing this train of investigation, it would already be an important step if it were only possible to ascertain in every case the quantity of urea produced during a state of fasting.

Now, although I do not imagine that the mere look at a table, which represents the results of carefully ascertained urine analyses, will of necessity forewarn us of the danger of impending uræmia, still I shall not omit to detail here the scanty data I have been able to collect as to the quantity of urea excreted in the urine during renal disease, by persons just before the advent of uræmic convulsions—remarking, at the same time, as I must, that the opportunities of making such observations in hospital patients are by no means large.

Far the larger number of cases which came into my Clinic and died with uræmic symptoms were persons who, up to the date of the attack, or within a short time of it, had been actively engaged in their occupations. For, as I have before noticed, uræmia, in my own experience, is both absolutely and relatively commoner among the very cases of renal disease which are not dropsical, and in persons, therefore, who are but little hindered from following their ordinary avocations. Over and over again it has happened to me to see apparently quite sound and sturdy looking people, who neither fancied they were ill themselves nor were thought ill by their friends, seized, in the midst of their work or at some place of amusement, with uræmic spasms, and quickly succumbing to their first attack of uræmia. And then, in spite of the aspect of good health, maintained vigor, and capital general nutrition of these patients, one finds after death the kidneys extensively degenerated.

---

[1] The ratio of urea production to each pound of body weight is clearly meant, though not precisely so stated in the text here.—TRANSLATOR'S NOTE.

The following analyses were instituted upon the urine furnished by patients suffering from renal disease, who were in the hospital, some shorter, some longer periods before the outbreak of uræmic convulsions.

C. K., a housemaid, twenty-eight years of age, had been under treatment for some months for albuminuria and bronchial catarrh; she was admitted into the hospital on account of an attack of endocarditis with high fever, on the 1st of March, 1867, and died of uræmic convulsions on the 1st of April. She was constantly feverish from the date of her admission. The mean of twenty analyses established that she passed a daily average of 762 c. ctm. of urine, and excreted 12.34 grms. of urea daily.

Post-mortem examination showed that she had contracted kidneys, hypertrophied left ventricle, and recent endocarditis implicating mitral valve.

Herr K. was treated in the hospital for twenty-seven months for dropsy, and left the institution entirely cured of his dropsy, but still passing albumen in his urine. Two months later he was admitted again highly anæmic and enfeebled, and tormented by constant vomiting. Stupor shortly set in, alternating with attacks of fearful depression of spirits. Twelve weeks after his admission he left the hospital, and died, a few days afterwards, at his home, with convulsions.

During his stay in the hospital a quantitative analysis was made of his urine, extending over twenty-five days. Upon an average he passed 1,018 c. ctm. of urine, containing 11.75 grms. of urea daily. The autopsy showed that there was no dropsy. Both kidneys were in a state of secondary contraction. Some considerable hypertrophy of left ventricle existed.

A youth, St., sixteen years of age, swollen so as to be quite shapeless with dropsy, four weeks after his reception into the hospital was attacked with epileptic spasms, which continued for three days, with such short intervals of remission that the patient never had time to recover his senses between the attacks. The convulsions then ceased. But six weeks later he experienced a fresh attack, of which he died. One thing worth noticing was, that during the interval of six weeks between the attacks, this patient took his food with a keen appetite, just as he was in the habit of doing before the first attack.

During the twenty-eight days of his residence in the hospital before he had the first epileptic seizure, he passed a daily average of 283 c. ctm. of urine of extraordinarily high specific gravity, and excreted upon a mean of eight analyses ten grms. of urea daily. In the course of the ensuing six weeks his urine, by reason of the diarrhœa which set in, could not be collected regularly. Still the average of the daily quantity, reckoned from thirty-five observations, gave 300 c. ctm. of urine as the quantity excreted per diem, and the mean of twelve separate examinations for urea, made during the same period, marked again ten grms. daily. The functional (quantitative) activity of the kidneys, therefore, remained about the same after the first attack of uræmia as before it, and yet in the mean time the cerebral functions had been restored to their full integrity. It is probable that constituents of the urine were removed from the economy by the copious diarrhœa which obtained. The young fellow remained as dropsical after the first severe attack of uræmia as he was before it. The kidneys were found to be greatly enlarged, yellow, and anæmic (chronic parenchymatous nephritis); *heart not hypertrophied;* the pia mater was in a state of gelatinous infiltration from œdema; a moderate amount of fluid in the ventricles, some of it too had escaped; brain substance remarkably anæmic, but firm, having throughout the well-marked polish due to moisture.

C. L., a maid-servant, twenty-two years old, was taken into the Clinic on account of dropsy and anasarca of moderate amount, which had existed only for a few days. Five days later she had two attacks of epileptiform spasms; death ensued directly upon the second attack. She had obstinate vomiting before this. During the six days she was under observation she passed 816 c. ctm. of urine daily on an average, and the mean of three analyses showed that 8.51 grms. of urea were excreted daily. The autopsy showed remarkable wasting of both kidneys; hypertrophy of the left ventricle.

It appears, from the above observations, that the outbreak of uræmic convulsions was in every instance preceded by a diminution in the excretion of urine, and especially of the urea, to a figure far below the ordinary mean average; the term *uræmic*, therefore, is correctly employed to denominate these symptoms. But the facts observed help us no further; they fail to explain why it is that uræmic symptoms are not forthcoming in cases conditioned under apparently quite identical circumstances. Repeatedly I have watched cases of contracted kidneys, with consecutive hypertrophy of the left ventricle, and with the daily excretion of urine and urea reduced to as low an ebb as in the cases I have narrated, advance to a fatal issue, without the occurrence of convulsions or protracted coma.

How, then, shall we explain the outbreak of epileptiform spasms in renal disease? We know of but one certain cause of epilepsy: namely, *the sudden interruption of the arterial blood* *supply to the brain.* Shall we accept this cause as operative in every attack of convulsions? I thought so once, because I had so often watched the commencement of an epileptic fit in persons who were subject to the habitual form of the disease, the attack in some instances coming on while I was in the act of conversing with them. In these cases a sudden pallor of the face was the first visible prelude to the commencing seizure. The assumption of an arterial spasm brought about by a vaso-motor centre would suffice of course to explain the whole affair. Nothnagel,[1] however, has already advanced some well-founded reasons for doubting the surmise, that the nerve-centre which produces the convulsions first receives its irritation from an anæmia produced by arterial spasm. His observations show that this pallor of the countenance at the commencement of a fit may be altogether absent. For example, both in the habitual form of epilepsy and in uræmia, we encounter cases where only certain groups of muscles twitch, and where the intelligence remains unaffected—cases which compel us to assume a purely local irritation confined to a very limited portion of the central motor nerve apparatus, and hence incompatible with the production of a general cerebral anæmia.

If we abstract our attention, however, from habitual epilepsy, we must recognize a whole series of causes, completely different, too, in their nature, which are capable of determining that complex of symptoms furnished in an epileptiform seizure. Thus at one time we find the first cause in disease of the brain and its membranes (in cerebro-spinal meningitis I have seen the most violent epileptic convulsions); at another, in some irritation of the peripheral nerves; in a third case it is some extreme overheating of the body, produced spontaneously by fever or through outside influences, by some artificial elevation of temperature;[2] or, lastly, it is some chemical substance introduced into the blood which thus provokes the attack. I lay some emphasis upon the cause last named, because I have twice witnessed epilepsy overtaking people after excessive intoxication with spirit, and these individuals were persons who had notoriously never had a fit before, nor, so far as I am aware, did they ever have another afterwards.

---

[1] Sammlung klin. Vorträge. No. 39.

[2] A stout, robust man, thirty-five years of age, went into a Russian steam-bath, heated to 53° R. (151° Fahr. = 66.5° Cent.), where I was engaged on some observations upon the temperature of his body. Within twenty minutes the thermometer placed in his rectum rose from 37.8° to 41.9° Centigrade, whereupon he had a violent convulsion and lost consciousness.

Surely we are not to assume—and certainly it has never been proven—that all these different conditions produce the epileptic convulsions in the same way, namely, by spasm of the blood-vessels ; and yet several of these occasional sources of epilepsy will fit in with the experiments [and theory] of Kussmaul and Tenner. One must remember, however, that in these experiments the chief importance is to be attached not to the anæmia of the brain, but to the insufficient supply of oxygen to this organ. Kussmaul and Tenner observed epileptiform twitchings occurring alike when they compressed the trachea of the animal experimented on, or when they tied all the arteries that supplied its brain.

Now it is possible to understand how the ingestion of enormous doses of alcohol may produce epileptiform spasms, if we bear in mind that the strong affinity of the oxygen in the blood for the alcohol must interfere with the distribution of such a quantity of this substance to the nerve elements in question as is requisite for the fulfilment of their normal functions. These nerve elements, furthermore, may be affected in exactly the same way when the urinary secretion is suppressed and nitrogenous excreta are retained in the circulation, the latter interfering with the processes of oxidation that are essential to these elements for the normal performance of their functions ; nay, I will even admit the possibility that a preternatural saturation of the same nerve-centres with water may entail exactly similar results. Unfortunately, this view does not admit of proof, and we must confess that, in the light of our present experience, the theory, that convulsive symptoms may also be provoked by either mechanical or thermic stimulation of the convulsive nerve-centre, has at least as much claim upon our acceptance.

The striking circumstance that uræmic convulsions come on in paroxysms, with intervals of perfect remission between them, the exciting cause remaining in undiminished force, is, physiologically, as little explicable as the periodicity of the attacks themselves in persons suffering from the habitual form of epilepsy.

I will not omit to state here that, with the exception of one single instance (vide Case VIII.), in every case of uræmic convulsions which I have seen, the temperature of the body, taken directly after the first attack, has been considerably elevated above the normal, being raised in two examples to 40.6° Cent. (105.1° Fahr.), and remaining thus abnormally high for some time, occasionally for days, when the convulsive attacks rapidly succeeded each other. These observations are opposed to the statements of Bourneville and Hervieux,[1] who claim that they observed a lowering rather than an elevation of the temperature of the body in uræmic convulsions. At all events, the temperature of the body in uræmia deserves far more attention than it has generally hitherto received. It is possible that through investigations in this direction some important light may be thrown upon the nature and causes of uræmia. I cannot think that the abnormal temperature which I observed could have resulted merely from the violent muscular exertions during the fits; it is the less likely, because the heat was maintained generally for hours beyond the duration of the actual spasms. Unfortunately I have no records of the temperature directly before the outbreak of the uræmic convulsions.

It is possible to imagine the sudden intrusion of some pernicious matter (noxa) into the system, which elevates the temperature after the way in which certain substances injected into the veins of an animal act as generators of inflammation. In any case, this elevation of temperature in uræmic convulsions cannot be referred to the formation of carbonate of ammonia in the blood, since the introduction of this salt into the circulation of animals, according to the unanimous results of experiments by Billroth, O. Weber, and, lastly, by Gosselin and Albert Robin,[2] is invariably followed by a lowering of the temperature of the body.

---

[1] Schmidt's Jahrbücher. Bd. 156, S. 297. 1872.

[2] L'urine ammoniacale et la fièvre urineuse. Archives Générales de Médecine. Mai, 1874, S. 530.

During uræmic coma the temperature may fall far below normal, and that, too, quite a long time before death.

Now the coma of uræmia, like its epileptiform convulsions, admits of explanation upon a variety of hypotheses. If a uræmic fit be followed by a comatose condition, this last may simply be interpreted as the final stage of this attack, such as is ordinarily observed after any severe epileptic attack. This coma, like that which follows an attack of the ordinary habitual form of epilepsy, may last a longer or shorter time, according to the gravity of the preceding seizure. Indeed there is but this difference to notice, namely, that the coma following the uræmia is ordinarily more protracted than that which succeeds the uncomplicated epileptic convulsion ; this is because the former are usually more violent than the latter, and follow each other with so much greater rapidity that the patients oftentimes do not recover their senses at all between the fits.

If, however, the comatose condition overtake a case of renal disease without the previous occurrence of convulsions, it may be often due to œdema of the brain and its membranes. At the same time I hold it to be an undesirable generalization to assume œdema as the explanation of the coma, and this because uræmic coma, like uræmic convulsions, is constantly observed in individuals who exhibit no trace of dropsy in any of their organs ; while anasarca, of the most extensive kind, may exist in other cases of gravest renal dropsy, and last for months, too, without there being any, even the very least, disturbance of the cerebral functions. I am also unwilling to accept the post-mortem appearances as absolute proofs of the pathological condition existing during the state of coma. When death is brought about by a protracted agony, be its cause what it may, one rarely fails to find a certain amount of serous infiltration of the brain. Now we may regard uræmic coma as a kind of prolonged death struggle. If, then, we adopt the hypothesis that uræmic coma is the result of a retention of nitrogenous excrementitious matters in the tissues of the body, we shall appreciate the aptness of the comparison made by Voit, who likened the condition to the extinguishment of a fire by the accumulation of its own ashes, or to suffocation from the non-elimination of the gaseous excretory products of respiration.

The view that the coma of chronic uræmia in many cases depends upon the retention of urinary materials in the blood and tissues, is favored by the circumstance that it is so often associated with that itching of the skin, previously noticed, which compels the patient to scratch himself incessantly, even when lying in a state of unconsciousness. I observed this symptom in two patients, who had crystals of urea upon the surface of their skins; but I have noticed itching oftener in those cases of chronic uræmia where there had been no convulsions, and in such especially as exhaled a urinous odor from their persons. This itching of the skin in uræmia I reckon an indisputable effect of the retention of urea or other urinary elements in the tissues, just as I hold the same event in icterus to be the result of the contamination of the nutrient fluids with biliary constituents, and the consequence of an impression made by these upon the terminal nerve filaments in the skin. This last opinion, I am sure, will hardly meet with any opposition, although not every case of icterus is accompanied by itching; and it is not every example of uræmia that excites this same symptom. And when further progress has been made in making analyses of the blood, we may, perhaps, also be able to state positively, *that it is not every case of uræmia that induces convulsions or coma.*

I am doubtful, however, whether the neuralgic symptoms occurring in such various nerve branches, which are described generally by the patient as rheumatic pains, and which we encounter so often in certain forms of renal disease, have any right to be attributed to the urinary constituents retained in the blood; I have noticed that they occur quite independently of any other uræmic symptoms, and have always supposed that they were due to the anæmia of the patient.

Among the results of the contamination of the blood, by urinary matters retained in it, are to be reckoned lastly *the marked tendency to inflammatory affections exhibited by pa-*

tients who have kidney disease. Here I am entirely in accord with the opinions expressed by Osborne and Traube, holding, as they do, the views which have been entertained and published by earlier writers who had not failed to notice with what frequency the so-called Bright's disease was complicated with inflammatory processes occurring in other organs. Indeed so common are they in renal disease, that, according to some, these inflammations are the immediate cause of death in the larger proportion of cases of Bright's disease, so-called; and probably they do lead to death, in the various forms of diffuse nephritis, more frequently than do dropsy, uræmia, and apoplexy reckoned together.

The peculiarity of these processes of inflammation in renal disease is the tendency which they manifest towards purulent exudation; the purulent infiltrations manifesting themselves most commonly as phlegmons of the subcutaneous connective tissue. Then again I have often seen erysipelatous inflammations of the skin occurring in renal cases; and, next to these, perhaps, soft infiltrations of the lungs are the commonest complications. Copious and at times purulent infiltrations frequently form in the pleural cavities and in the pericardium; more rarely in the peritoneum and in the meshes of the pia mater. Once only have I seen purulent effusion take place into several joints; the case was one of chronic parenchymatous nephritis connected with amyloid degeneration. The occurrence of these inflammatory processes in the most varied parts of the body is certainly not dependent upon the previous existence of dropsy, since, so far as my experience goes, they occur more frequently in the cases where there has either never been any dropsy, or where, at all events, this did not exist at the date of the inflammation. This peculiar proclivity of the tissues to become inflamed cannot be the result of a watery condition of the blood serum, or it would happen more especially in the dropsical forms of the disease; and, further, we know that hydræmia due to other causes entails no particular predisposition to inflammatory processes.

Moreover, as to the localization of the exudation in any particular case, we shall find that this is determined by the particular state of the organ assailed. Here, too, we may call attention to the relative frequency of pericardial exudations in cases of hypertrophied left ventricle brought about by the contracted state of the kidneys, and still more especially to the great frequency and danger of purulent phlegmonous inflammations occurring in the anasarcous limbs, in the scrotum, in the labia pudenda, and in the face, in cases of renal disease with dropsy.

Death by gangrene is the ordinary issue of these phlegmonous inflammations.

3. *Symptoms due to Disturbances of the General Nutrition produced by Renal Disease.*

Renal affections influence the general nutrition of the body in a variety of ways. *In the first place, in most cases of renal disease, the general nutrition suffers in consequence of the losses of albumen inflicted upon the organism; in some cases there is also a loss of the formed constituents of the blood.* It is true enough that this loss of serum albumen has been asserted to be of small importance to the general nutrition of the body, this view being based on the assumption that the serum albumen is something quite different from the organ albumen—in fact, a substance that plays no direct part in the nutrition of the different organs. But, as Voit very justly remarks: "Whatever view we may entertain in regard to the use to which albumen is put in the economy, one thing we can be sure of, namely, the aggregate mass of working cells and the total supply of oxygen—upon which, in turn, depends the activity of the processes of retrograde metamorphosis, or, in other words, the functional activity—are determined by this element."

Now, if we consult clinical experience, we shall find that renal diseases—apart from their disturbing influence upon the organs of digestion, or from the wasting effects of any febrile movement that may be associated with them, or from their diverting, like cancer, the nourishing material to heteroplastic uses—produce their injurious effects upon the general nutrition—namely, anæmia, emaciation, and weakness—all the more quickly, in proportion to the abundance of the loss of albumen which the organism experiences in consequence of the albuminuria which accompanies these renal diseases. In the case of a diffuse nephritis it is not necessary that the excretion of formed elements of the blood should be very abundant in order that there should be produced a pretty high degree of anæmia and weakness in the course of a very short time.

How much the muscles waste and the subcutaneous fat disappears, commensurately with the albumen that drains away in the urine, and how quickly the natural plumpness and rotundity of the form are lost, are changes which are apt to escape our observation, owing to the presence of dropsical swelling of the general subcutaneous tissue. The wasting, however, becomes apparent for a time, when, from profuse sweating or other similar drain, a sudden absorption of the dropsy takes place.

On the other hand, genuine contracting kidney disease may have been progressing for years, involving a very trifling loss of albumen, although leading to considerable atrophy of the renal glands, and yet the normal color of the skin and mucous membranes need not have altered, or the plumpness of the form or the corporeal activity have been really impaired. But I shall treat of the relations of the albumen losses to the different forms of diffuse nephritis hereafter, when I enter more minutely into the description of these affections.

Kidney affections, in the next place, very often do harm to the general nutrition, through the various functional disorders of the digestive apparatus to which they give rise. Dyspepsia, vomiting, and diarrhœa are such frequent and ordinary symptoms of renal disease that the mere fact of their repeated concurrence proves some causal relation between it and them. Still, it is certain that these symptoms owe their immediate etiology to very different causes.

Thus the *dyspepsia* is in some cases manifestly only the result of the extreme anæmia provoked by the renal disease, and, like that produced by all other anæmic states, is simply to be referred to an insufficiency of the secretion from the peptic glands of the stomach; and, at the autopsy, the mucous membrane of the stomach, beyond exhibiting extreme emptiness of its blood-vessels, shows no appreciable pathological alteration. But in other cases considerable œdema of the mucous membrane is discovered at the post-mortem examination, which must have prevented the glands from furnishing a normal secretion. Finally, the contents of the stomach are sometimes found to be highly ammoniacal, showing that urea has made its way out of the blood into the secretions of the stomach, and has accumulated in its interior and been transformed into carbonate of ammonia, which, neutralizing the acid reaction of the gastric juices, has deprived these of their digestive virtue. The dyspepsia due to this last source, however, is generally brought about only a very short time before death.

The *vomiting* of renal disease, like the dyspepsia, owes its origin to a variety of causes. In every case of acute swelling of the kidney, or of violent inflammation of the pelvis or of the ureter of one of these organs, one may look upon the attendant vomiting as a strictly reflex act; it is started by an irritant impression made upon the nerves—either those supplied to the capsules of the kidneys, and therefore implicated in the tension to which these organs when swollen would be subjected, or those which run to the pelvis of the gland, and which may either be involved themselves in the process of inflammation or have suffered directly from the same cause (a calculus, for example) that produced this.

In much the same way vomiting may succeed an inflammatory irritation of the peritoneal coat of the kidney, as in nephritic abscess, or in the course of the development of new growths, or after abnormal distention of the pelvis of the organ by pyelitis or hydronephrosis. In all these instances the vomiting is attended by pain of more or less acute kind, and ordinarily paroxysmal in its nature—pain which subsides in one class of cases with subsidence of the swelling, and in the other with the abatement of the sharp irritation provoked by the calculus in the pelvis of the kidney, although likely in the last-mentioned example to return with renewed violence over and over again.

The vomiting of renal disease may also be due to œdema of the mucous membrane of the stomach. In the cases of this nature which I have seen, the vomiting took place, as a rule, only in the morning, before breakfast, when a quantity of watery fluid of very low specific gravity (1002), and possessed of a faintly acid reaction, was rejected; the appetite the while remaining tolerably good, and such meals as were taken being digested without further difficulty. I assumed that the vomiting in these cases was due to the œdema of the gastric mucous membrane, because the individuals affected by it were dropsical, and I could discover neither urinary constituents nor their derivatives in the fluids vomited. At a later date, and in a case where this water-brash persisted up to the very last, I had the opportunity of confirming my suspicions at the autopsy. It appears likely that the fluids effused beneath the mucous membrane of the stomach transude into its cavity, and by their mere volume excite the vomiting. But it astonished me to find that these patients retained their appetite; and in two cases of mine even large meals have been regularly digested. My observations, however, entirely accord with what Bernard and Barreswill noticed in the animals they nephrotomized, and in which the secretion of gastric juice took place in abundance a very few hours after the operation, the juice itself, in spite of being mixed with some ammoniacal salts, still retaining its acid reaction and digestive capacity.

Still, it cannot be denied that in many cases of renal disease, vomiting, as an obstinate and irrepressible feature thereof, follows upon the retention of urinary materials in the blood, and is then symptomatic of uræmia. In many instances it is the first symptom of chronic uræmia and the precursor of severe nervous manifestations, convulsions, and coma.

This uræmic vomiting produces serious effects upon the general state of nutrition, interfering, as it does, with the assimilation of food already ingested, and usually causing it to be rejected almost as quickly as it is swallowed. Besides, it is frequently found associated with the greatest possible dislike to all solid articles of food, and especially to meat; and it is on this account that one notices a rapid deterioration of the strength from the moment that this troublesome and unconquerable symptom appears. This form of vomiting, too, at its commencement ordinarily sets in only in the morning upon first awaking, but after a while occurs also after any meal and at any time. At first the vomit still presents an acid reaction, and many observers have found urea in it, although I have not, as yet, succeeded in finding any. Later on in the disease, when the patients generally abstain from nourishment altogether, the fluids vomited are alkaline, and may have a penetrating ammoniacal odor, or, if mixed with blood—a thing not at all so rare—may emit a disgusting foul odor, which also communicates itself to the breath.

That the ammonia contained in the vomit in these cases originates from the urea thrown out of the blood and decomposed in the interior of the stomach, does not admit of dispute, after all we have learned from clinical observers (who have demonstrated the presence of urea in the vomit), and from the experiments of Bernard and Barreswill ; and it is equally certain that this ammonia, directly its quantity suffices to neutralize the acidity of the gastric juices, must completely nullify the process of digestion. But it still appears to me not wholly settled whether this form of vomiting, which I, from its invariable connection with other uræmic symptoms, have specified as uræmic, ought from first to last to be regarded as the result of an irritation of the nerves of the stomach by abnormal gastric contents. Doubtless this view of the case is correct when large quantities of volatile ammonia compounds have been formed in this organ, so that its contents present a strong alkaline reaction, and blood has issued from its mucous membrane in consequence of it. Still, so long as the contents of the stomach retain their acid character, and the digestive power of the gastric juice remains, and so long as the vomiting and allied sensation of morning sickness (the stomach therefore, at this time, containing no food) are corrected upon the first taking of food at breakfast—as Christison reports, and as I too have oftentimes observed—it is still possible to refer the vomiting to direct irritation, through the impure state of the blood, of those nerve-centres which co-ordinate the act of vomiting. The question has hitherto been scarcely mooted, but it is one that I am unable at the present time to solve.

Diarrhœa also may arise as a complication of renal disease, and, though it occurs more rarely than vomiting, it may prove quite as obstinate and may hasten the deterioration of the patient's strength quite as decidedly. This loss of strength is due to the rapidity with which the intestines are emptied, whereby the absorption of the food taken is prevented. I have often been able to demonstrate notable quantities of peptones in the loose stools passed by persons suffering with renal disease, as well as in those passed by persons suffering from other diseases, when, in spite of their diarrhœa, they still continue to take an abundance of food.

Then, again, the diarrhœa in renal cases may arise from a variety of causes, quite apart, of course, from that process of ulceration in the intestines which occurs so very frequently as a complication of the amyloid degeneration of the kidney.

In the first place, the diarrhœa may be due to œdema of the mucous membrane of the intestines, and then occurs only in the dropsical forms of renal disease. In these cases it comes on as a rule in the form of short, sharp attacks, the stools being abundant, very watery, and but slightly colored; and in this way sometimes the dropsy is either greatly reduced or completely removed. This last event I only witnessed once, and, just as the dropsy disappeared, a uræmic attack came on, which terminated life. Occasionally the diarrhœa of patients with renal dropsy,—that looseness of the bowels which I attribute to œdema of the intestines,—becomes most obstinate, and then one finds mucus in abundance, portions of pus and traces of blood mixed up with the evacuations; but we are not on that account justified in arriving at the conclusion that we shall find, after death, the œdematous mucous membrane already in a state of ulceration. Thus in the case of a boy, sixteen years of age, who had parenchymatous nephritis, and was excessively dropsical, I observed a profuse diarrhœa, which lasted for several weeks, and caused his rectum, shortly before death, to remain constantly prolapsed; and yet in this case, although an enormous quantity of purulent matter escaped from the bowel, the mucous lining of the intestines, and especially that of the colon, was found, after death, beyond being pale and highly œdematous, perfectly intact. The so-called secondary dysentery, which has been referred to by several writers as a common result of renal disease, I have never seen.

But even diarrhœa may be a symptom of uræmia, in so far as it can be produced by the passage of urinary constituents into the intestinal canal and their conversion into carbonate of ammonia. In such cases I have recognized the strong ammoniacal odor of the fæces, and have been able to show chemically the large amount of ammonia present in them ; but the diarrhœa has then been contemporaneous with vomiting of ammoniacal matters, and has constituted, in the cases I have seen, practically one of the final symptoms.

Lastly, in some cases of renal disease a condition of things arises which, more perhaps than any other which has been hitherto described, hastens and favors the decline of the general nutrition and strength of the patient. I refer to a *hemorrhagic diathesis.* Petechial spots appear upon the skin, and besides these the predisposition to bleed is evinced by spontaneous epistaxis, by hemorrhages that take place both from the stomach and from the intestines, by bleedings from the mucous membrane of the mouth, and by hæmoptysis. In fact, up to the present time the only mucous tract from which I have observed no tendency to bleed has been that of the genito-urinary apparatus of women ; the commonest source of all is the nasal mucous membrane. After death we are unable to discover the slightest anatomical lesion to explain these oftentimes profuse bleedings, which must take place per diapedesin.

It is only when this hemorrhage takes the form of epistaxis that it may occur repeatedly during a prolonged period, and ought not, therefore, to be accepted as an immediate harbinger of death ; when derived from other sources and accompanied by petechiæ, it has invariably indicated the near approach of dissolution, and this although the patient at the time of its first occurrence may have been feeling pretty well and comfortable. Indeed in every example of well-established hemorrhagic cachexia that I have met with, the patients whom it has befallen have not survived two weeks from the commencement of the bleedings. They often lose blood simultaneously from different mucous tracts—from the lungs and intestines, from the mouth and nose, and from the stomach and bowels together.

Hemorrhages of the kind which I have described have invariably occurred only in cases of genuine contracting kidney (granular atrophy), and never, so far as I have seen, in dropsical persons. Only one individual, a housemaid, twenty-two years of age, whom I attended, exhibited some slight anasarca of the face and extremities; but she vomited large masses of blood, although there was not the slightest lesion of the mucous membrane of her stomach to be discovered after death.

Opinions are divided as to the exact cause of these hemorrhages. While some (Lecorché,[1] for example, who withal describes this symptom as an ordinary precursor of uræmia and dependent upon atheroma of the arteries, a condition of things which is not encountered under these circumstances) consider that increase of tension in the arterial system is enough to explain it, others, as Gosselin and Robin,[2] regard it as a sign of ammoniæmia. This view they believed themselves justified in holding, because they saw bleeding occur from the noses of guinea-pigs when carbonate of ammonia solution had been injected into their veins; and the carbonate of ammonia, which they think provokes these hemorrhages, in cases of renal disease, they derive from the urea with which the blood is loaded. My own observations establish neither the one theory nor the other, and they also fail to confirm the idea that these hemorrhages are due to either excessive thinness of the blood or atheromatous degeneration of the arteries. Traube, too, is undecided whether to attribute the profuse epistaxis he has seen in cases of contracted kidney with hypertrophied left ventricle, to the constituents of the urine retained in the blood—and if so, whether in its etiology it can be considered as having a parallel in the profuse hemorrhages incident to icteric affections—or to refer them to increased arterial tension.[3] But I have known the hemorrhages to continue up to the very last, whereas the heart's power had sunk to a low ebb already several days before death, the pulse being soft and empty; and in this case the blood drawn five hours before death by bleeding from a vein, and tested by the method of Kühne and Strauch, showed that there was not a trace of ammonia present. The specific gravity of the blood as a whole, estimated by the pyknometer, marked 1050, that of the blood serum being 1030.58. The water content of the serum was 866.90 per 1,000, the solids 133.10 per 1,000, the albumen 82.81. The full details of this case are appended.

---

[1] **Archives Générales de Médecine. April, 1874.**

[2] **Eodem loco. Mai, 1874.**

[3] ***Traube*, Gesammelte Beiträge zur Pathologie und Physiologie. II. S. 741.**

CASE VIII.—Hermann St., laborer from Neumühlen, thirty-two years of age, was taken into the Medical Clinic on Nov. 11, 1873. According to his account, he suffered, in 1864, for several months with some chest affection, and since then he had frequently been indisposed and disinclined to work. During the past few years, especially, he had noticed an increased thirst, as well as a desire to pass his water more frequently than usual. He never was a spirit drinker.

Condition on Admission.—Of medium height; stoutly built, with a moderate amount of fat about him; skin sallow; capillary networks of cheek and nose highly varicose and blue, standing out through the skin; slight œdema over ankles of both legs; expression heavy and languid.

Thorax.—Lungs everywhere contain air; coarse mucous râles heard throughout the bronchi; heart's apex-beat in fifth intercostal space in the line of the nipple; area of cardiac dullness slightly extended beyond normal limits in every direction. Heart sounds clear, yet an accentuation of the mitral sound with the systole, and of the aortic with the diastole, is distinctly perceptible; pulse at the wrist soft, rather empty, 72; temperature normal; appetite small; sleep, according to the patient's statement, uneasy, broken by disagreeable dreams. Urine passed in rather large quantity, of a very light straw-yellow color; specific gravity 1010; contains a rather large quantity of albumen and a good many casts, some of which are narrow and hyaline, others thick, dark, and granular. (For general summary of the urinary excretion see below.)

The *diagnosis* made was that of genuine contracting kidney, with secondary hypertrophy of the left ventricle, although no intensification of the heart's impulse or increase of tension in the arteries could be positively affirmed to exist. I was not surprised at this, however, as my previous experience had taught me that, when general debility sets in, the symptoms which ordinarily appoint hypertrophy of the left ventricle may disappear entirely.

Treatment.—Thirty-one grains of iodide of iron, in thirteen drachms of bitter tincture[1]:—thirty drops to be taken thrice daily after eating. To remain in bed.

By the 13th November, after persistent profuse diuresis, the anasarca of the ankles had entirely disappeared. During three consecutive nights, from the 16th inst., the patient had severe attacks of dyspnœa, which compelled him to sit upright in bed for some hours. Loud sibilant sounds proceeded from the bronchi, and were heard all over the ward; but there was no cough, no expectoration, and no evidence furnished by the stethoscope of any accumulation of fluid in the bronchi;

---

[1] A tincture of gentian, European centaury, zedoary and orange berries. *German Pharm.*

there was also no alteration of the resonance upon percussion over the lungs; these were pure asthmatic attacks, therefore. In the meantime, however, the vigor of the heart's contractions had greatly increased; the pulse was full and tense, although not so characteristically jerking as it ordinarily is in hypertrophy of the left ventricle. The urinary excretion had also increased during the same time, upon an average of five examinations, to above 3,000 c. ctm. daily.

On the fourth night the attack of asthma did not come on; but the patient woke, on the morning of the 20th of November, with headache, from which he had often suffered previously; when he got up to take his breakfast, he vomited, without experiencing any previous sensation of sickness, some watery stuff having an acid reaction but in which no urea could be detected.

From the 20th of November to the 17th of December, the day of this patient's death, the vomiting was daily repeated, with few exceptions; at first it occurred only early in the morning, but later on many times in the course of the day. Repeated examinations of this acid fluid thus rejected by the stomach for urea invariably gave negative results. All our efforts to stop this vomiting—broken ice, tincture of iodine, creosote, opiates—were all without avail; at the same time, the patient's strength visibly failed, his appetite having rapidly disappeared altogether. His temperature, taken in the rectum, often fell below 37° C. (98.6° Fahr.), and it was remarkable that, on an average, it marked a half or a whole degree higher on the days when there was no vomiting. The pulse, notwithstanding the repeated vomiting, always retained its strength.

On the night between the 6th and the 7th of December there was a considerable loss of blood per anum; the rectum, which was examined, furnished no explanation of this. And now ensued a daily loss of blood from the same source. On the 9th of December an additional loss took place, though less in quantity, from the nose and mouth. Various punctiform ecchymoses, of about the size of a split pea, appeared upon the mucous membrane of the gums, from which blood constantly oozed. The skin surface remained free from petechiæ, and the urine, which continued to be abundant (1,800 c. ctm. daily, upon an average of three measurements), contained no blood. The patient's collapsed condition increased, the heart began to fail, the pulse becoming small and feeble, and the temperature remained almost constantly at 37° C. (98.6° Fahr.).

Ordered: wine, and hypodermic injections of camphorated oil (camphor, a grain and a half, dissolved in fifteen minims of olive oil at a dose).

On December 14th there was retention of urine. After an interval of thirty-two hours, during which no urine had been voided by the patient, a catheter was passed, and 1,040 c. ctm. of urine were drawn off, the fluid presenting a specific gravity of 1012 and containing 1.5 per cent. of urea. The vomiting and hemorrhages persisted.

On December 15th spasmodic twitchings took place in various groups of muscles, and from time to time the patient's whole frame was shaken as by an electric shock. The intellect was affected; after long consideration patient answered questions slowly. Dec. 16th.—The twitchings were both stronger and more frequent.

December 17th.—Could not pass his water, although wishing to do so; this morning, 125 c. ctm. were drawn off by catheter. Repeated vomitings occurred, a dark-brown looking material being thrown up, which presented an alkaline reaction and had a strong ammoniacal odor. Frequent spasmodic twitchings set in, principally affecting the muscles of the neck, so that the head was jerked first to the left and then to the right. There was nearly as much twitching of the muscles of the upper extremities, but less of the legs and trunk. Patient unconscious. Temperature, taken in the rectum, 37.4° C. (99° Fahr.). At half-past eleven he was bled from the median vein of the left arm to the amount of 125 c. ctm. of blood. Death ensued four and a half hours later, after a protracted agony. The following table gives the analysis of the blood, drawn five hours before death :

Specific gravity of the blood as a whole (estimated by the pyknometer)	1050
Specific gravity of the blood serum (estimated by the pyknometer)	1030.58
In 1000 parts of blood serum the contained water was	866.90
" " " " " " solids were	183.10
" " " " " " albumen was	82.81

No carbonate of ammonia could be distinguished in the blood by the method of Kühne and Strauch. The unusual density of the blood serum was doubtless due to the fact that there had been an excessive secretion of water by the kidneys up to the very last, while only a small amount of water had been ingested.

From November 13th up to the date when the hemorrhages began, the 6th of December, this patient's urine was carefully collected and the daily quantity estimated. The mean average of twenty-three such measurements gave 2,470 c. ctm. as the mean daily quantity. For seven days out of these twenty-three, the percentage, as well as the absolute quantity of urea contained in the urine per diem, was taken. These seven analyses showed that up to the date of the hemorrhages the patient excreted a mean daily average of 26.6 grms. of urea. And that this rate corresponds pretty exactly with the mean average quantity of urea excreted daily throughout the time the patient lay under our observation, is shown by the little variation which took place in the specific gravity of the urine excreted each day, which fluctuated only between 1009 and 1011, and by the equally slight variation in the daily percentage quantity of urea for the six days during which it was estimated—namely, between 1.0 and 1.1 per cent. It was only on the 5th of December that the percentage amount of urea contained in the urine rose as high as 1.3 per cent.

From the date of the commencement of the hemorrhages, December 6th, it became impossible, by reason of the patient's weakness and his frequent intestinal evacuations, to collect the entire urine separately. From the 14th to the 17th of December (the date of death) the urine was drawn off by a catheter, and, reckoned altogether, the total quantity was 1,165 c. ctm., containing 17.60 grms. of urea, or, taking these three last days separately, a mean average for each day of 388 c. ctm. and 5.87 grms. of urea.

I append the account of the post-mortem examination from the words of my colleague, Heller.

Skin somewhat muddy looking; scarcely a trace left of œdema in lower extremities; muscles strongly developed; both lungs free from attachments; a few drops of clear serum in the pleural cavities; lungs on removal collapsed very little, being highly emphysematous in their anterior parts; the pleura covering the upper lobe of the right lung presented a little recent fibrinous deposit upon it. This upper lobe of right lung was anteriorly bloodless and pretty markedly œdematous, although still containing air in it; but its posterior half contained no air, and was the seat of gray-red infiltration unequally distributed through its substance, thus in some parts being more yellow, in others of a darker gray-red color; it was granular on section. Middle lobe contained air throughout its substance, but was bloodless and highly œdematous; lower lobe contained less air, was highly œdematous and fuller of blood; left lung contained air, but was œdematous. Pericardium held a few drops of clear serum; heart rather large, 12½ ctm. long, 11 ctm. broad; ventricles firmly contracted, and but little blood-clot in the auricles; valves normal; muscular substance firm and dry; the left conus arteriosus measured 2.2 ctm., the right 0.4 ctm. in thickness. Only a few drops of serum in the peritoneal cavity. Left kidney very small, only measuring 9.5 ctm. in length, 5.4 ctm. in breadth, and 2.2 ctm. in thickness; capsule everywhere firmly adherent; surface, throughout, both finely and coarsely granular, and of a pale-yellow color, with here and there a few spots of stellate capillary injection. The cortical substance appeared on section of a very pale-yellow color, and was only 7 mm. broad; the pyramids were very small, and of a pale gray-red color; the walls of the renal artery were soft. The right kidney was just like the left, only considerably smaller than it—8.3 ctm. long, 4.3 ctm. broad, and 2.5 ctm. thick. The right renal artery was normal. The stomach contained a pretty large quantity of highly bile-stained fluid, which smelt very strongly of ammonia; its mucous membrane was tinged of a deep slate-color. Small intestine moderately filled, containing large quantities of chyme of a dark grayish-green color, which in the upper portions was much mixed with gall, and smelt very strongly of ammonia. Mucous membrane throughout slate-colored, moderately injected with blood, and presenting some ecchymoses. Dark tarry masses in cæcum; its mucous membrane in some parts strongly injected, but, deeper down, of a pale color, with gray-green masses of fæces present; rectum everywhere slate colored, and containing fæces coated with some tenacious mucus. Skull-cap not symmetrically shaped; its surface somewhat uneven; substance compact, very heavy. Firm blood—and fibrin—coagula in longitudinal sinus; dura mater thin and bloodless. Inner membranes very bloodless, throughout of delicate consistence, and only in the sulci, and principally then in the posterior parts, slightly cloudy and œdematous. Brain substance moderately bloodless, firm, easily separated from its membranes; lateral ventricles moderately distended with clear serum; central ganglia pretty full of blood; cerebellum rather soft and anæmic. There were a few connective tissue adhesions to the membranes at the base of the brain. The walls of the arteries were thin and they were well filled.

The autopsy, then, furnished no information upon the exact etiology of the hemorrhages which had taken place at the same time from different tracts of mucous membrane, tracts removed so widely apart one from the other. The case simply teaches us that the bleedings in this particular instance were due neither to ammonia in the blood nor to the extreme watery condition of this fluid, nor even, lastly, to any excessive tension in the arterial system.

I have known similar multiple hemorrhages to occur in patients suffering from renal disease where none of the ordinary uræmic symptoms were exhibited.

THE DIFFUSE DISEASES OF THE KIDNEYS.

Historical Notice.

The knowledge which medical men possess of the diffuse diseases of the kidneys, their symptomatology and their effects upon the organism, is, comparatively speaking, new. Apart from a few accounts of diseased states of the kidney found scattered through older literature, and records of appearances occasionally found at post-mortem examinations, and independently of notices of the appearance of blood in the urine, an occurrence which even the earlier physicians had observed in the urine of persons whose kidneys were in some way diseased; apart from the clinically recognized fact that dropsy may manifest itself in persons suffering from disease of the kidneys, and the discovery of Cotugno, that the urine of dropsical subjects sometimes contains albumen,—it cannot be said that the physicians of past centuries possessed any real knowledge of the diffuse affections of the kidney, although these were of such frequent occurrence; they were not able either to diagnose them in the living subject, or to measure their progress, and, finally, they did not appreciate the danger to which patients suffering from renal disease are exposed.

It was reserved for Richard Bright to first explore this important field of pathological research. Bright, having recognized certain structural changes in the kidneys taken from the dead body, was the first person to show that the appearance of albumen in the secretion furnished by these glands, might be accepted as evidence of these alterations; and he even recognized the causal relationship between affections of the kidney and other symptoms of disease, such as dropsy, in particular, hypertrophy of the left ventricle of the heart, and a variety of other disorders of the nervous functions. Bright also estimated the annual number of deaths, due to renal disease and occurring in London alone, at 500.

But this historical introduction to my description of the diffuse diseases of the kidneys would, in truth, expand to bounds far beyond what is becoming the nature of this work, were I to relate here every bit of intimation that lies broadcast through ancient literature in reference to these diseases, or to notice every opportunely narrated surmise in this direction that might be discovered. Nor is this necessary, for the eminent P. Rayer has taken the trouble to collect together all the old—dare I call them the prehistoric—records that bear upon this subject, and has appended them to the conclusion of his writings upon the affections of the kidney, which he describes together under the name of "néphrite albumineuse" (second volume of Traité des Maladies des Reins).

If now we date the history of these affections back only to the time when we began to gain some knowledge of their pathological anatomy, their symptoms and sequelæ, it may be said that this began only when Bright published his first memoir upon this subject; and Christison remarks with perfect justice, in the preface to his pamphlet on the Granular Degeneration of the Kidneys, "that neither a few previous chance observations nor some still rarer obscure and in themselves incomplete deductions can be considered of sufficient importance to deprive this first discovery of the value which it has proved to medicine."

In the year 1827 Dr. Richard Bright published the first volume of his Reports on Medical Cases; and the first chapter of these reports has the heading, "Cases illustrative of some of the Appearances on the Examination of Diseases terminating in Dropsical Effusion." Then, in the first part of this same chapter, the cases of disease are described under the title, "Diseased Kidney in Dropsy," prefaced by some general remarks upon the various causes which can give rise to dropsy. Bright remarks, that up to that time too little attention had been paid to the structural changes of the kidneys as causes of dropsy, and adds that in those cases which owe their origin to disease of the kidneys he had often found the urine coagulable by heat, a property never manifested by the urine in dropsy due to liver affections. On the other hand, he never failed to find alterations in the structure of the kidneys in the bodies of persons in whom death had been preceded by dropsy and albuminuria.

The causes of these structural changes in the kidneys consisted, as Bright believed, in certain injurious influences, which, acting upon either the stomach or the skin, disturbed the functions of the kidneys, either by disordering the circulation through them, or by bringing about a state of decided inflammation in them.

In one category he placed cases of acute dropsy with albuminous urine, which began mostly by hæmaturia, and were the result of exposure to cold; in the other he set cases which happened to individuals who had brought themselves down by irregular living and the abuse of alcohol. The urine derived from persons belonging to the latter category was, he found, often turbid from the presence of saline precipitates, which, however, redissolved upon the application of a gentle heat; but here the kidneys were always found in a decidedly degenerate condition, whereas in the first set of cases he sometimes encountered instances in which these organs were merely hyperæmic—"gorged with blood."

Bright appends some general remarks to his report of these twenty-three cases of dropsy with albuminuria, described in his treatise, and seventeen of which came to post-mortem examination. His observations led him to distinguish three varieties, if not three quite totally different forms, of structural disease of the kidney, each one of which was associated with the excretion of albuminous urine.

His *first form* appeared to him to consist of a state of degeneration, which might be described at its commencement as a mere enfeebled condition of the organ. The kidney loses its wonted firmness, and obtains a yellow-spotted aspect, and, on section, presents this clear yellow coloration, intermixed with gray, throughout its entire cortical substance. The gland remains nearly of normal size, but its pyramids look paler than is natural. This form is encountered most often in cachectic individuals—phthisical persons, for example. Bright could discover no morbid deposits in these kidneys. In the advanced stages of this form of disease, the organ acquires a tuberculated aspect, the elevated knobs on it being paler than the surrounding parts, and not admitting any injection fluid into the arteries supplying them.

The *second form* is distinguished by the granular condition which characterized the entire cortical substance, and is produced by an abundant interstitial exudation of an opaque substance. At first this change in the kidney substance is only recognizable as an accentuation of the natural spotty appearance of the organ, which looks as if fine grains of sand had been deposited in its substance. The kidney also seems less firm than normal, and with the advance of the disease these deposits throughout the cortical substance become more and more distinct and more numerous, so that at last the entire surface of the gland appears slightly roughened. The kidney is sometimes larger, sometimes smaller than normal, and at times it presents externally nearly the same nodular appearance that is presented by the last stage of the first form.

In his *third form* the entire surface of the kidney is roughened and uneven, being covered with a countless multitude of small nodules, the size of a pin-head, some yellow, some red, and some bluish colored. The organ thus obtains a lobulated appearance, is hard, and cuts with as much resistance as fibro-cartilage; the pyramids are crowded closer together and approach nearer to the surface than they should; in a word, every part of the organ appears contracted ("it appears, in short, like a contraction of every part of the organ"), and the amount of interstitial deposit appears to be less than in the second form. The pervading color is a red gray, sometimes of a deeper, at others of a lighter hue. In most of these cases the urine is highly albuminous, although, in one instance, boiling it only led to the precipitation of a dense, branny sediment of brown color.

At the same time, Bright does not pin his faith very positively upon the varieties which he has given out as distinct forms;

nay, he is rather inclined to allow that his first form may never pass beyond its first stage, and that what he has reckoned as later stages of its development may really be a state of things more properly appertaining to his second form—in fact, he is doubtful whether his second and third forms ought not to be regarded as mere modifications of each other, or as more or less advanced stages of one and the same process of disease.

But Bright also recognized other states of the kidney, connected with albuminuria, of a transitory nature, which came and disappeared again from day to day. One of the pathological conditions leading to this kind of albuminuria he describes as "a preternatural softness of the kidney;" while another, according to him, consisted in a blocking up of the tubuli uriniferi with white sedimentary concretions having the aspect of tiny coagula. Associated with the former of these conditions of the kidney, he had noticed a corresponding loss of firmness in the liver, the spleen, and the heart; to which last he attributed the weakness of the heart's action, noticed as a symptom during the patient's life. In the cases where the tubules were blocked up, he found the kidneys firmer than natural. Bright, therefore, had no doubt that we should come to recognize many other conditions of the kidneys as sources of albuminuria.

In several of the cases observed by Bright, and reported in his treatise, the chemical constitution of the urine and the blood was ascertained by his friend, John Bostock, and the results of these analyses were incorporated into the work. Indeed it is quite astonishing to notice the complete manner in which he surveyed nearly every important question which could be raised by his discovery. He pursued the same subject in later observations, the results of which were published by him in the second part of his Reports of Medical Cases, in 1831, and again later on in Guy's Hospital Reports, in 1836, 1840, and 1843.

Meanwhile Bright's discovery had excited the attention of hospital physicians in several of the principal towns of Great Britain. Christison had already in 1829 published, in the Edinburgh Medical and Surgical Review, the results of his observations in the new field of discovery, the article being entitled

"Observations on the Variety of Dropsy which depends on Diseased Kidney." He was followed, in 1831, by his friend and hospital colleague, Dr. James Gregory, who published a treatise in the same journal, with the title, "On Diseased States of the Kidneys, connected during Life with Albuminous Urine." Then, in the year 1834, Dr. Osborne, of Dublin, wrote a work "On the Nature and Treatment of Dropsies, accompanied by Coagulable Urine and Suppressed Perspiration," which appeared in the Dublin Journal of Medical and Chemical Science. Still, even in 1838, Christison complains, in the preface to his memoir upon Granular Degeneration of the Kidneys and its Connection with Dropsy, Inflammation, and other Maladies (a work translated by Johann Mayer, and published, with notes, by Carl Rokitansky, in Vienna, 1841), that Guy's Hospital and the Edinburgh Infirmary were, up to that date, the only institutions in Great Britain that could be said to have contributed to the enlargement of this department of pathology.

The writers above named concurred entirely in the doctrine developed by Bright, namely, that the principal symptoms observed during life—dropsy and albuminuria are here especially intended—are due to pathological processes taking place in the kidneys, and to the resulting structural alterations in these organs; but they advanced no more decided opinions than Bright had upon the character of these pathological processes or upon the nature of the changes which they produced in the kidneys.

Nevertheless exception was shortly taken to this doctrine of Bright, that dropsy and albuminuria arose from some organic change in the kidneys.

Dr. Elliotson,[1] of London, while admitting that albumen usually appeared in the urinary secretion during these alterations of the kidneys which Bright had described, called attention to a large number of cases which had fallen under his own observation, in which complete recovery, both from the dropsy and from the albuminuria, had taken place; and he contended, therefore, against the essential dependence of these symptoms upon the changes observed in these glands. So far as the pathological signification of albuminuria was concerned. Elliotson maintained that it derived its importance far less from the quantity of albumen contained in the urine, and from the condition of the kidney, whatever one might interpret this to be, than from the general state of the whole system, of which these particular symptoms only afforded outward evidence.

---

[1] Clinical Lectures on Dropsy. London Medical Gazette. 1830.

Graves,[1] who did not dispute the fact of the urine being albuminous in these altered states of the kidney described by Bright, was unwilling to accept this writer's interpretation of their etiology. He regarded the albuminuria as the cause, and the pathological changes in the kidney as the result of it. "In dropsy," he says, "one perceives that there is an inclination towards a superfluous excretion of albuminous fluid throughout the body, in the kidneys as well as in every other part; but since this excretion takes place in the kidneys in the extremely fine tubes of their cortical substance, and is there mixed with urinary salts and various acids, one can hardly wonder if the albumen molecules thus shed should remain as coagula in the secreting tubes, block them up, gradually distend them, and after this way affect that obstruction of this glandular tissue to which the name of Bright's disease has been attached."

In opposition to this objection, Bright maintained his own standpoint, namely, that in the disease which he had described the mainly important item was the kidney change; and besides his own Edinburgh colleagues, some, at all events, of the foreign writers, in France especially Rayer, sided with him. A number of other English and French writers, on the other hand, favored Graves' view. This very same difference of views upon the relation borne by the excretion of albuminous urine to the anatomical changes in the kidneys, first described by Bright, still prevails even at the present time. Each view, from the beginning, has had its different advocates, and continues to have them still, although each special pleader has certainly introduced numerous modifications into his case, according to his individual conception of the different arguments to be pleaded.

Prout, who was among the adherents to the humoro-pathological view of the case, taught that the albuminous substances of the blood, whenever, through fever or any other cause, they were rendered unfit for assimilation, would be excreted by the kidneys. Owen Rees and Malmsten likewise regarded the blood-change as the primary one ; their belief was founded partly upon the perceptible departure from its normal condition, which the blood ordinarily exhibited in renal diseases, although they failed to show that in point of time this anomalous state of the blood preceded the renal changes, and partly upon the circumstance that albumen was often discovered in the urine during life, while after death the kidneys which had furnished it presented no departure from the normal condition. Even Valentin,[1] who was the first person to occupy himself with the microscopical examination of diseased kidneys, thought his investigations afforded him further reasons for referring renal disease to antecedent blood disease; he attributed the kidney changes to the precipitation of albumen from the urine. The albuminuria, however, he considered a symptom of a general disease, whose essential feature resided in the excretion of an abnormal quantity of albumen from the blood, the urine in these cases being the means by which its elimination was effected.

---

[1] London Medical Gazette. Dec., 1831.

Robin[2] referred the albuminuria to some imperfect elaboration of the albuminous substances in the blood, and attributed this to an incomplete oxidation within the blood-vessels.

Gubler[3] explained the albuminuria as the result of an absolute or relative excess of albumen in the blood—a condition, according to him, which could be brought about by the nature of the food, by errors of digestion in the primæ viæ, or by disorders of the liver, or of the respiratory organs, or of the nutritional processes in the tissues (the processes of assimilation and retrograde metamorphosis). Gubler, moreover, was not the first person to give expression to the idea that functional disorder of the liver might be a cause of albuminuria. Graves had asserted the same thing before him.

---

[1] Repertorium für Anatomie und Physiologie, 1837, S. 290, ff., und folgender Jahrgang.

[2] Des causes du passage de l'albumine dans les urines. Comptes Rendus de l'Academie des Sciences. 1851.

[3] Communicated in an unpublished lecture by Gubler upon albuminuria, and referred to by Jaccoud in some remarks appended by him to his translation of Graves' Clinical Lectures (Leçons de Clinique Médicale de R. J. Graves, traduit par le Dr. Jaccoud. Paris, 1863. Tome II. p. 397).

Jaccoud, finally, in his Paris thesis, "Des Conditions Pathogéniques de l'Albuminurie,'' undertook to establish the doctrine of albuminuria in the sense of its being a blood disease. The above-named writers of the humoral school of pathology were all entirely agreed in this, that the first cause of every albuminuria lay in some blood change, and that this preceded and produced the structural alterations of the kidney. The proofs of this vague assumption, however, were entirely wanting, and nearly every one of its supporters established the cause of this presumed alteration of the blood upon some different foundation, either on disorder of the digestive or the respiratory function, or on arrest of the cutaneous functions. Jaccoud held that Gubler's theory was the only one arrived at by direct experiment, although, from what is told us, it is impossible to discover what is meant by Gubler's absolute and relative excess of albumen in the blood; neither are we informed that Gubler ever demonstrated his presumed excess of albumen to obtain in renal disease.

In the previously mentioned remarks appended to his translation of Graves' lectures, Jaccoud sums up the results of some observations he had made upon two individuals affected with albuminuria; one of them, as the autopsy proved, had granular atrophy of the kidneys, while the other removed himself from observation before the termination of his case. Now, in the fæces of both these patients Jaccoud found albumen, although the intestinal mucous membrane, examined in the body of the first named, was found to be perfectly normal; further, the cerebrospinal fluid taken from the same case contained albumen in notable quantity. But from these facts Jaccoud deduces the broadest possible conclusions. "It is plain,'' he says, "that throughout the whole course of the symptoms the changes in the kidney are of only secondary consideration; it is further apparent that it is the state of the entire system which is, above all else, the exciting cause of these functional disturbances, since albumen does not escape alone from the diseased kidneys, but also from the extensive surface of an entirely sound mucous membrane. Hence it follows that we are not to accept albuminuria as pathological evidence of this or that disease of the kidney, but as the visible and tangible token of some general disorder, whose grave import and incurability stand in direct ratio to the duration, and beyond all else to the extent (généralisation) of this abnormal phenomenon" [*i. e.*, the draining away of albumen from various sources.—TRANS.].

It is impossible to say that this high-sounding phraseology of Jaccoud is much clearer or more convincing than the ideas put forward by his predecessors, of whose lack of clearness he himself complains.

While, in the study of the morbid conditions described by Bright, one set of writers started with the assumption that some error of blood formation must be at their foundation, and addressed their attention primarily to the symptom of albuminuria, paying little or no heed to what was going on in the kidney, and being themselves much more concerned to demonstrate that the anatomical lesions of these organs are entirely subordinate and secondary to the albuminuria, another set, on the other hand, were very ready to consider the presence of albumen in the urine as an infallible evidence of renal disease. Bright, it is true, endeavored to guard his doctrine from such an interpretation, but yet it is difficult to draw any other meaning from his publications than that albumen may appear in the urine at a time when the kidney structures are still perfectly sound, but that its occurrence is to be interpreted as indicating the beginning of structural changes. These changes, as Bright thought, could be made, by the skilful interposition of the physician, to recede and give place again to the normal condition; in other instances, however, they were certain to lead, sooner or later, to that condition of the kidneys first described by him—a condition admitting of no complete repair, and which establishes a disease which, sooner or later, will of necessity lead to death, either by dropsy, or by inflammation of the serous membranes of the lungs, or by grave cerebral symptoms.

Meanwhile those who, adhering to the doctrine of Bright, held that the renal malady was the starting-point of the entire train of symptoms involved in the disease, directed their chief attention to the pathological changes in the kidneys. A most careful work in this direction was shortly issued by P. Rayer (Traité des Maladies des Reins, published in two vols., Paris, 1840). Although those who had preceded him had not ventured to express a positive opinion upon the nature and essence of those changes undergone by the kidneys, and hitherto only named after Bright, Rayer did not hesitate to describe them as inflammatory, and to group them together under the name of albuminous nephritis. "La néphrite," he says,[1] "est principalement caractérisée, pendant la vie, par la présence d'une quantité notable d'albumine, avec ou sans globules sanguins dans l'urine, par une moindre proportion des sels et de l'urée dans ce liquide, dont la pesanteur spécifique est presque toujours plus faible que dans l'état sain; enfin, par la coïncidence ou le developpement ultérieur d'une hydropisie particulière du tissu cellulaire et des membranes séreuses. La néphrite albumineuse peut être aiguë ou chronique, febrile ou apyrétique."

These several states of the kidney, therefore, Rayer groups together as if they constituted manifestations of a single disease; and yet, at the same time, in his description of their anatomical characters, he is compelled to distinguish six different forms of his albuminous nephritis—forms, too, which he by no means invariably treats as various stages or phases of development of one and the same process of disease. He does not enter into the relations which exist between his anatomical varieties and the pathology and symptomatology of the disease, further than to state that the two first of the forms distinguished by him correspond to acute, and all the rest to chronic nephritis.

Superabundant as the materials are which Rayer has collected in his excellent work, and carefully as he has investigated the etiological relations obtaining between the diffuse renal diseases, on the one hand, and other morbid processes with their after effects, on the other, he serves up the anatomico-pathological portion of his work in a most bald and scanty manner. He derives his six forms of albuminous nephritis from different peculiarities in the external aspect of the kidneys, and only after he has got well on in his work does he make mention of the relation of his several varieties to their producing causes, or state under what influences these alterations of the kidney are brought about. In his pathological investigations Rayer was still restricted to naked-eye observations, although, before the appearance of his work, Valentin had already, with the help of the microscope, endeavored to arrive more accurately at the anatomical changes which the kidneys underwent in chronic albuminuria. Meanwhile to Rayer's important influence may be ascribed the fact that the inflammatory nature of all these pathological states of the kidney, which Bright had described, became pretty generally recognized, and that the first thorough pathologico-histological work, which emanated from the pen of Reinhardt, and was published in 1850, in the first volume of the Annals of the Charité Hospital in Berlin, was also based on this doctrine.

---

[1] l. c. p 97.

As a result of his histological investigations, Reinhardt pointed out that the entire group of pathological states of the kidney discovered by Bright were the results of inflammation; and to this inflammation he attached the name of diffuse nephritis, because of its general extension throughout the affected organ; but he showed that it was an inflammation which might pursue various courses, according to the causes that induced it and the constitution of the individual whom it attacked; and he explained "that the entire state of things collectively described as Bright's disease belongs to no one single process of disease, but ought rather to be understood as localizations of entirely distinct processes of disease that take place in the kidneys, and which are best comprehended under the name of diffuse nephritis."

It was in 1851, the following year, that Frerichs published his well-known monograph,[1] giving the results of his own contemporaneously pursued inquiries. In the minute histological description which he gives of those diseased states of the kidney which are associated with the excretion of albuminous urine and the extrusion of casts, one perceives that the two investigators are pretty nearly in accord. Both are agreed that the renal changes proceed in certain stages, which are distinct, and follow one upon the other, and each distinguishes three such stages: *a first, of hyperæmia; a second, of exudation with fatty degeneration of epithelium; and a third, of new-growth* (hyperplasia) *of connective tissue with atrophy of the organ as its ultimate issue.*

---

[1] Die Bright'sche Nierenkrankheit und deren Behandlung. Braunschweig, 1851.

The term inflammation, however, for designating this process of disease, did not satisfy Frerichs, who thought that the word did not define the proceeding with sufficient distinctness; he preferred, therefore, to employ the customary name of *Bright's disease* for it, as one at all events historically justified.

Both the above-named German investigators stoutly maintain the identity of the various pathological states of the kidney described by Bright and his followers, although admitting that these may originate in different ways, progress under different conditions, and therefore possibly be accompanied by very distinct trains of symptoms. Both, too, are agreed in this, that the multiplicity of the anatomical lesions, presented by the kidney in Bright's disease, "form one uninterrupted chain, from hyperæmia and fatty degeneration down to atrophy of the organ,—a chain of events whose separate links, we shall perceive, are closely welded together, directly we have learnt for ourselves the proportional parts, which the intensity of the process of exudation—always a fluctuating item—and the metamorphosis of its products—a constantly advancing one—play in the proceeding."[1]

It was not long, however, before objections were raised from various quarters against the identity of these different morbid changes in the kidney, thus grouped together under the title of Bright's disease.

Traube,[2] upon the authority of his own minute anatomical investigations, asserted that the changes produced in the kidneys by hyperæmia, due to passive congestion, although associated with albuminuria and the extrusion of fibrinous casts from the tubes of Bellini, could not be reckoned amongst the results of Bright's disease.

---

[1] *Frerichs*, l. c. S. 172.

[2] Ueber den Zusammenhang von Herz und Nierenkrankeiten. Berlin, 1856.

Bamberger[1] boldly endeavored to include the kidney changes brought about by venous congestion among the other forms of Bright's disease, and to maintain its identity with them. It was enough for his purpose, without further reasoning, that cylindrical casts should form in the renal tubules of kidneys which were the seat of passive congestion; for at that time the exudation of fibrin from the blood-vessels was pretty universally accepted as infallible evidence of inflammation, although Traube had already, it is true, contended against this conclusion.

Since then this question has been decided both by clinical observations and by pathological research; and the opinion first broached by Traube, as to the distinction which exists between passive congestion of the kidneys and its consequences, and the states described as Bright's disease, has been generally acknowledged to be a perfectly correct one.

The pathological anatomists had already, at an earlier date, recognized and named a condition of the kidneys, which was entirely distinct from those changes usually designated as Bright's disease, but which had nevertheless hitherto been included among these. Rokitansky, in the first edition of his text-book of Pathological Anatomy, 1842, was the first person to describe the lardaceous kidney as the last one of the eight forms into which he divided Bright's disease. Meckel next, in 1853, demonstrated that in lardaceous degeneration, wherever this occurred, whether in the kidney, the liver, or the spleen, a peculiar substance was present that gave a particular color reaction with iodine and sulphuric acid. Virchow, and many others after him, subjected this substance to further observation, and investigated more particularly the conditions under which this degeneration takes place in the larger abdominal organs. Traube was the first to indicate how this *amyloid affection of the kidney*, as it was called by Virchow, could be diagnosed and distinguished clinically from other renal diseases; and since then this form of affection, although usually attended by both dropsy and albuminuria, has been generally accepted as a process quite distinct from Bright's disease, as regards both its morbid anatomy and its clinical details. In the meantime, both in France and in Germany, the idea entertained of Bright's disease was, that it was some uniform and special process applicable to a whole series of pathological conditions, as to the exact mode of origin and development of which people accepted the views laid down by Reinhardt and Frerichs.

---

[1] Ueber die Beziehungen zwischen Morbus Brightii und Herzkrankheiten. Virchow's Archiv. Bd. 11, S. 12.

Anticipating, however, both Reinhardt and Frerichs, Dr. George Johnson, of London, published, in a series of memoirs, his views upon Bright's disease, showing that this affection, which had heretofore been considered to exist in one form only, existed in a variety of forms; according to him it was not merely necessary to distinguish an acute and a chronic form, but several different kinds of disease, each one of which differed essentially from the others. This author put forward his opinions, in detail, in a large work, On the Diseases of the Kidneys, published in 1852; and in this, besides waxy degeneration, he distinguished an acute and a chronic desquamative nephritis, a non-desquamative form of disease, and a fatty degeneration of the kidney; next to these he made out a granular fatty kidney, distinct from the last named, and a mottled form of fatty kidney; but he supposed that his non-desquamative form subsequently passed into fatty degeneration. Dr. Johnson endeavored to base his classification of the diffuse diseases of the kidneys upon the results of his investigations into the minute anatomy of the diseased kidneys. These investigations are of importance in one particular, namely, they were the first that took cognizance of those changes which the walls of the blood-vessels undergo in the disease in question. His observations have, in this respect, proved the precursors of those subsequent researches which have once more been prominently pushed forward by his own countrymen, namely, Grainger Stewart, Gull, and others,—investigations which at the present time appear likely to play an important rôle in the pathology of renal diseases. Johnson's investigations were especially directed to the changes that take place in the epithelium of the renal tubules, and he omits all notice of the inter-tubular connective tissue.

Johnson's acute desquamative nephritis corresponds with the first stage of Bright's disease, as conceived by Reinhardt and Frerichs. His non-desquamative form and his fatty degeneration of the kidney (which latter must not be confounded with the fatty kidney as conceived at the present day) agree, at least in part, with the second stage made by the same writers; while his chronic desquamative nephritis accords entirely with Reinhardt and Frerichs' third stage, that of atrophy, when the kidney exhibits a general process of contraction, which arises through shedding, without previous swelling, of the epithelium of the tubuli uriniferi, with subsequent complete destruction of the tubes. In the course of this process of contraction, a hyperplastic thickening takes place, according to Johnson, in the smallest arteries of the kidney.

It should be stated, however, that Johnson looks at matters throughout from a humoro-pathological point of view, and seems to have no hesitation in explaining pathological processes as existing for this or that particular (useful) purpose. He thinks that the contamination of the blood with pernicious matters is the one real cause of all these kidney diseases. The system, he believes, is engaged in an endeavor to eliminate these substances from itself, and to this end selects the route through the kidneys; the effort of the epithelium of the renal tubes to carry out this object leads to its becoming damaged, and finally to its destruction; for the cells are engaged not merely in accomplishing their normal secretory function, but in striving to excrete the abnormal constituents which have entered into the blood.

It was only in England that Johnson's views at first found any adherents.

In Germany they were first thoroughly sifted by Virchow, both in his Lectures,[1] in 1847, and in his well-known pamphlet, "Ueber parenchymatöse Entzündung," which appeared in 1852. In this treatise Virchow, after acknowledging the inflammatory nature of the kidney changes named after Bright, complains that the name "Bright's disease" has been given, on the one hand, to all those changes which eventually terminate in granular degeneration of the kidneys, even if the process should run its course in a chronic form, without dropsy, albuminuria, or ostensible symptoms of uræmia; and on the other, to all those conditions where albuminuria chances to arise in some slight departure of the kidney from its normal state, but one which brings neither granular degeneration nor dropsy in its train.

---

[1] Virchow's Archiv. Bd. 4, S. 260.

As one of the slighter forms, Virchow points to that catarrhal inflammation of the renal tubes which follows the application of irritating substances, like cantharides, blisters, and mustard poultices to the skin. The catarrhal inflammation thus originated, first affects the urinary outlets, and subsequently extends backwards to the papillæ and into the straight tubules. A similar state of affairs is seen in the kidney affection following cholera. "In catarrh of the renal tubules the chief change consists, first, in an increase of the number of the cells themselves, and further on, in a later stage, in an alteration of the cells, which become first more granular-looking and opaque, and afterwards present an irregular, broken outline and yellowish-gray aspect;"—this is Johnson's acute desquamative nephritis. The catarrh of the renal tubules may advance to croupous inflammation, *i. e.*, a fibrinous exudation may appear in the tubules. This croup, then, of the tubuli uriniferi is, in some measure, a graver sort or higher grade of catarrhal inflammation. Furthermore, the croupous exudations may extend up even to the Malpighian bodies.

Virchow, lastly, distinguishes a parenchymatous inflammation of the kidneys, his third form, and thus describes it: "This consists essentially in an alteration of the epithelial cells, and principally, of course, of those which lie nearest to the Malpighian bodies in the curling tubes, in the more intricate or cross parts of the tubuli uriniferi." Virchow, as is well known, mentions as one of the characteristics of parenchymatous inflammation, that the inflammatory exudation is taken up by the elements that compose the actual tissues,—in parenchymatous nephritis, therefore, by the epithelial cells of the tubuli. "These cells therefore swell, become cloudy, are less transparent and more granular, and are, at the same time, often more friable than they should be. Two issues are now possible: the epithelial elements either soften and smelt down into a pulpy detritus (protein jelly), or else pass into fatty metamorphosis, and finally form an emulsified milky or creamy pap." Virchow, having previously insisted that inflammation with parenchymatous exudation did not exclude the simultaneous occurrence of interstitial and of free exudation—nay, showing as he did, that all these three processes of exudation could proceed together,—only further refers to this combination of affairs, in his description of parenchymatous nephritis, to remark, that catarrhal, croupous, and parenchymatous nephritis do not uncommonly occur simultaneously,—adding, further, that it is just this combined complication which is calculated to produce the highest degree of degeneration of the kidney; and for this complex condition he advises us to reserve the name of Bright's disease, if the term is to be employed at all, out of grateful acknowledgment to Dr. Bright himself. Certainly, the slighter affections, whether catarrhal or croupous, do not entail complete degeneration of the kidneys, but this follows as the result of the occurrence of parenchymatous changes.

Beer,[1] in a lengthy work, directs attention principally to the changes that take place in the interstitial connective substance in the diffuse affections of the kidneys.

This was the work to which Traube[2] appealed in 1860, in his demand that the name of Bright's disease might be given up, since it comprehended within itself four distinct processes of disease, namely: 1st, the changes of the kidney produced by venous stasis (or congestion); 2d, amyloid degeneration; 3d and 4th, two forms of diffuse or interstitial nephritis—one of which forms, the *circumcapsular*, was anatomically characterized by development of connective tissue chiefly round about the glomeruli; and the other, the *intertubular*, by new growth of tissue principally between the tubes of Bellini. Then, besides, the clinical aspect of these two forms is different: the former, the circumcapsular nephritis, pursues, as it appears, a chronic course; in it the urine soon exhibits a yellow color and a low specific gravity, and, apart from its more or less abundant sediment, resembles that furnished by the contracting kidney; while the latter, the second form, begins with hæmaturia. When contraction of the kidney supervenes, the train of symptoms which characterizes both these forms of nephritis and that which characterizes the amyloid degeneration become identical.

---

[1] *Beer*, Die Bindesubstanz der Menschlichen Niere. Berlin, 1859.

[2] Zur Pathologie der Nierenkrankheiten. Gesammelte Beiträge zur Pathologie und Physiologie. 2. Bd. 2. Abtheilung, S. 966.

The changes which take place in the epithelium, according to Traube, are indisputably of a secondary nature, and the conception previously formed of parenchymatous nephritis must therefore be abandoned as untenable. Traube holds fast to this opinion in opposition to Rosenstein, who asserts that the fatty degeneration that results from processes of inflammation ought to be distinguished from that which constitutes a simple retrograde metamorphosis preceded by no inflammatory process. It is impossible, as Traube believes, by examining fattily degenerated epithelium, to decide whether the degeneration has succeeded inflammation or some other promoting cause. Swelling alone, he says, is no evidence of inflammation; the outcome of inflammation is not swelling merely, but the multiplication or proliferation of elemental parts.

Later on, Traube[1] proposes the name of *nephritis hemorrhagica* for his inter-tubular nephritis—insisting, however, in the main, upon the views previously expressed by him regarding the nature of the anatomical changes. Traube's hemorrhagic nephritis, therefore, corresponds with the first stage, and in some cases with the second stage, of the morbus Brightii of other writers, and his capsular nephritis with the third stage.

Rosenstein[2] holds firmly to the more restricted idea entertained by the older writers upon morbus Brightii, and separates it from Virchow's catarrhal nephritis as well as from the changes impressed upon the kidneys by states of congestion and from amyloid degeneration. He describes the anatomical course of the disease as consisting of three stages, after Reinhardt and Frerichs' scheme, and names them *diffuse nephritis*, *parenchymatous nephritis*, and *granular degeneration of the kidneys*, the latter being the result of parenchymatous nephritis. Rosenstein mentions, as the essential feature of this process, the changes brought about by parenchymatous inflammation (using this term in the sense attached to it by Virchow), with its ultimate destruction of the epithelium, and insists upon the circumstance that the final result, atrophy of the kidney, may be reached without the interstitial tissue being in the least degree implicated, simply in consequence of the decay of the epithelium. "But" (to quote his own words) "this atrophy is for the most part associated with some changes of the interstitial connective tissue, consisting at one time in fibrillar, at another in cellular hyperplasia, whose spontaneous shrinking assists in producing contraction of the organ."

---

[1] l. c. S. 1029.

[2] Die Pathologie und Therapie der Nierenkrankheiten. Zweite Auflage. Berlin, 1870.

Virchow, in his Cellular Pathology, again speaks of Bright's disease, and mentions particularly how important it is, among the various conditions of the kidney to which the name Bright's disease has heretofore been given, to distinguish whether the changes start from the vessels (amyloid degeneration), from the epithelium (parenchymatous nephritis), or from the interstitial tissue (the indurative form). "At the same time, however, it must not be overlooked," he says, "that these three different forms do not always occur as well-defined varieties, clearly distinct each from the other; it is more apt to be the case that two, or even all three of these forms occur at the same time and in the same kidney, and that one form of disease may have lasted a long while, and yet end at last by being complicated by one of the others, or by both of them together. The order in which they are most apt to be associated is the following: to a simple parenchymatous or interstitial nephritis of old standing, there is joined, in the stage of marasmus, amyloid degeneration."

This opinion of Virchow's upon the nature of the various forms of chronic diffuse kidney affections has been adopted in its essential features by Grainger Stewart,[1] and forms the groundwork of his exposition of Bright's disease. On the title of his work, Bright's diseases are made to figure in the plural number. In his text he distinguishes, as quite distinct forms of disease, inflammation, waxy degeneration, and shrinking of the kidneys, the last form being termed by him "cirrhosis of the kidney." At the same time he admits that atrophy of the kidneys may also be associated with the two first-named forms as a third stage. This secondary atrophy, however, may be distinguished from the primary cirrhotic shrinking of the organ by the circumstance that in the cirrhotic form the wasting is accomplished by proliferation and subsequent contraction of the interstitial connective tissue, the epithelium of the urine tubes vanishing under the compression of the growing tissue; whereas in the parenchymatous form of inflammation, on the other hand, a primary fatty degeneration and destruction of the epithelium (precedes and) is the cause of the atrophy, and in the amyloid degeneration, the epithelium dies *in situ* (by necrobiosis), in consequence of the disease of the blood-vessels. Wherefore, in the cirrhotic atrophied kidney, the interstitial tissue is absolutely increased in quantity; while in both the other forms of atrophy it is increased only relatively, and merely in the measure in which the volume of the rest of the glandular substance is diminished by the destruction of the epithelial cells. Grainger Stewart, too, assumes that amyloid degeneration may co-exist with parenchymatous inflammation, and cirrhosis of the kidney with parenchymatous inflammation.

---

[1] A Practical Treatise on Bright's Disease of the Kidneys. 2d edition. Edinburgh, 1871.

Grainger Stewart departs in one particular from Virchow's older views, since he places catarrhal and croupous nephritis as the first stage of parenchymatous nephritis.

Other English writers, and especially Dickinson, treat of the diffuse diseases of the kidneys very much upon the same plan as that adopted by Grainger Stewart.

Lecorché,[1] among French writers, principally follows the views of Dickinson and Grainger Stewart, although in some few points he differs from them; thus he refers those changes, which result from venous congestion, back again to interstitial nephritis or sclerosis of the kidneys—Grainger Stewart's cirrhosis. But Lecorché's views have hitherto only been known to me through some papers of his which appeared in the Archives Générales de Médecine. Indeed I came across his large work, cited above by me, for the first time while I was employed in the compilation of this historical introduction. Still I shall have the opportunity of referring to this author again in my text.

---

[1] Traité des Maladies des Reins. Paris, 1875.

The contracted kidney has recently become the subject of special pathological study on the part of Sir W. W. Gull and Henry G. Sutton.[1] While former writers, and not a few modern ones, as already stated, regard the diffuse renal affections as the result and evidence of some presupposed deterioration of the blood, and others, on the other hand, look upon the kidney changes as purely local affections, explaining certain more or less constant concomitant alterations in other organs of the body as consequences of the renal malady, Gull and Sutton put forward the opinion that the form of kidney atrophy named after Bright is merely a part symptom of a widespread general affection. The essential nature of this systemic affection they define to be a hyperplastic growth of the connective tissue sheath of the arteries, and of the connective tissue immediately surrounding the capillaries, and this they call *arterio-capillary fibrosis.* This periarteritis may develop throughout the entire arterial system, but is found more commonly and is best established in some particular organs—as, for instance, in the arteries of the kidneys, the pia mater, the retina, the heart, the lungs, the stomach, the spleen, and the skin. These authors would have us designate this periarteritis universalis by the term morbus Brightii; and they thus arrive at the following paradoxical conclusion, that *morbus Brightii may exist without implication of the kidneys in the process.*

But I will reserve the minuter examination of the doctrine put forward by Gull and Sutton, upon the nature of genuine atrophy of the kidney, for its appropriate section in this work, and will only remark here that both these authors reject Johnson's views, according to whom the kidney disease is the result of a deteriorated blood. They point to old age as the principal original cause of this disease in the walls of the vessels; but in this respect their views appear to have led them, finally, into the error of confounding the changes brought about by age throughout the system with a process that is indisputably specific. At the same time, as they themselves inform us, they witnessed an extensive grade of contraction in the kidney of a boy of only nine years, and again in a case between sixteen and twenty; they also observed this condition of the kidney in two cases between twenty and thirty, and in ten between thirty and forty.

---

[1] **On the Pathology of the Morbid State, commonly called Chronic Bright's Disease, with Contracted Kidney (Arterio-Capillary Fibrosis). Med.-Chir. Trans. 2d Series. Vol. 37. Entire Series. Vol. 55.**

Furthermore, they do not attribute the symptoms observed in the course of this renal disease, as they have hitherto been referred by clinical physicians, to the particular state of the kidneys, and to the resulting disturbance of their functions, but seek to explain them upon the ground of these assumed changes in the vessels and consequently in the nutrition of the affected organs. Thus the headache would be due to periarteritis of the meninges, the dyspeptic difficulties to a corresponding state of the vessels in the mucous membrane of the stomach, the dryness of the skin to alterations of the blood-vessels in the general covering of the body, etc.

Kelsch[1] has quite recently issued a critique upon the doctrine of morbus Brightii, the grounds for his objections being his own pathological inquiries. He protests against the misuse which the German histologists have made of their microscopes, whereby they have been leading the laborers in this department of pathology into error for years past. Bright never expressed himself positively upon the nature of the disease which he had discovered. Rayer was the first to express a decided opinion in favor of its inflammatory nature. An advance, says Kelsch; for certain forms of it certainly are inflammatory; but still an error, for all are not; and, for these last, white degeneration and granular degeneration were better terms. Kelsch especially disallows the applicability of the term inflammation, when attached to Virchow's parenchymatous nephritis. What Virchow understood by parenchymatous inflammation, is, according to Kelsch, only anæmic necrosis, in which the vessels and interstitial tissue of the kidney play no part at all ; and in contending against this idea of parenchymatous inflammation, he employs the same arguments which Traube did before him, and in the same sense. He denies, however, Traube's assumption that an inflammatory change (cellular infiltration) occurs in the interstitial connective tissue in the pure forms of the affection described by Virchow as parenchymatous nephritis, although conceding as a rare occurrence the (occasional) combination of the latter form with interstitial nephritis. The term nephritis is, according to Kelsch, alone admissible when attached to the form of disease known as contracted kidney (Grainger Stewart's cirrhosis, Lecorché's sclerose), since in that condition a new formation of tissue actually does take place. The first stage of interstitial nephritis begins in the form of a deposition of embryonal cells in the interstitial tissue, sometimes with swelling of the entire organ ; the next stage is characterized by the organization of these embryonal cells into connective tissue, and ends with atrophy of the organ. The bringing together of the different forms of diffuse kidney diseases under the name of a single morbid process having three stages, is a purely artificial and arbitrary affair. The first stage recognized by different authors is entirely evolved out of their imagination, and rests on no anatomical observations whatsoever ; while the second stage has nothing whatever in common with inflammation. Its characteristic feature is a primary degeneration of the epithelium. This second stage (the large white kidney, as it is called in England) is the result of insufficient nutrition supplied to the epithelium, which suffers in consequence retrograde metamorphosis. This state of kidney, therefore, only arises in individuals who are very much run down by disease, whether phthisis, scrofulosis, syphilis, or some bone affection. It is associated, often, with amyloid degeneration of the vessels, and is very rarely complicated by interstitial nephritis. As a rule, no trace of inflammation is discoverable, and the capillaries and interstitial tissues remain perfectly intact. There is also no indication whatever of a proliferation of the epithelial structures, which ought to have been forthcoming, if inflammation had been the cause of their degeneration.

---

[1] **Revue Critique et Recherches Anatomo-pathologiques sur la Maladie de Bright. Archives de Physiologie Normale et Pathologique. Deuxième Série. Tome I. Paris, 1874, p. 722.**

The kidney malady which succeeds scarlet fever is a true interstitial nephritis, as is that too which follows small-pox, and the changes in the epithelium which take place in these diseases, are strictly secondary processes, signifying merely the death of the epithelium.

The changes which characterize the cholera kidney are entirely different from those which are observed in inflammation ; an acute necrosis of cells takes place, the vessels and interstitial structures remaining intact. This necrosis of cells takes place in the cortical, and not, as Virchow said, in the pyramidal portions. Virchow's catarrhal and croupous varieties of nephritis do not exist. Kelsch, at the same time, admits that the epithelium shed after an attack of cholera may be replaced by a new growth of epithelium, and says he has seen this himself ; the young cells colored brightly with carmine.

Kelsch separates the congestive kidney from nephritis. The changes are confined to the epithelium, and especially to that of the curling tubuli. The connective tissue remains unaltered.

It is apparent, from all that has been stated above, that the doctrine of the diffuse renal diseases cannot be regarded as settled, from any point of view. The pathological anatomists are not agreed in their interpretation of the nature of the process under discussion, and are still far from being in accord upon the identity or non-identity of certain of the conditions. What Rindfleisch already complained of, in the first edition of his Compendium of Pathological Histology (Leipzig, 1867, S. 416), holds good down to the present date : " The pathological anatomy of inflammation of the kidney is certainly the subject which has stimulated the most investigation, and yet to-day it is the least complete chapter of the whole work." Small wonder, then, that up to this time so few practitioners should clearly apprehend or understand the wide variety of symptoms exhibited in the course of these kidney diseases.

Such being the state of things, I am well aware that it may seem presumptuous in me to step forward with a new treatise in this special department, where the pathology and, unfortunately to all concerned, the therapeutics too are encompassed with difficulties and full of so much obscurity. It is true that I have enjoyed ample opportunities of watching the complaint, in the course of twenty-five years' active practice, both in hospital and in private, in a country where renal disease is exceptionally common. I might also state that, in the beginning of my career as a physician, I was Frerichs' assistant in the medical wards of this hospital, and that during this period his work on Morbus Brightii was published, a work that marked a special epoch in the history of this disease. These, as a matter of course, were all favoring circumstances, but yet they were not sufficient to justify me in entering upon such an undertaking.

The question I had to ask myself was this, Is there a real need of a new work upon a subject which has been so laboriously worked at down to the very latest date? To this question it seemed to me that I could give a most positive answer in the affirmative. A clinical experience, extending over many years, and an extensive intercourse with my brother practitioners, have taught me that the ideas at the present time prevailing among the greater number of German physicians upon the nature of the affections hitherto comprehended under the term Bright's disease, represent anything but correct views of the events which actually take place in these processes; indeed many practitioners do not possess a right estimate of the situation in particular cases, and are unable to utilize the signs furnished by the disease for the purposes of prognosis and treatment.

Carrying on my observations from a purely clinical standpoint, I have been led to separate the diffuse renal affections attended by albuminuria into several essentially distinct pathological processes, and have earnestly striven to recognize at the bedside the peculiarities which attach to each one of these different processes, in the hope of thereby securing a firm basis on which the diagnosis, prognosis, and—so far as possible—the treatment of these different affections might be established. As early as 1871 I published the results of my experience, and the views I entertained upon the pathology of the diffuse renal diseases, in a short work;[1] and it is no small encouragement to me now, in my present undertaking, to find that the results to which clinical observations had led me have been substantially confirmed at the hands of so many pathologists who have occupied themselves in the minute study of the diseased processes in the kidney more than I have done.

As to what I have now to say in the matter, I may confess that I do not come forward with the idea that I shall be able to cope with all the difficulties that will beset my path, but with the intention that my work may further some advances, clear up some obscurities, and thus at all events be of some use to general practitioners. This intention I am content to plead in justification of my treatise, however far its fulfilment may fall short of its author's endeavor.

The brief period accorded me for composing this work, and the disagreeable interruptions encountered in its accomplishment, have made it impossible for me to make use in its entirety of the abundant literature that exists upon my subject; indeed, for that matter, I should not have been able even to procure all that has been published on this topic. I have confined myself, in the above short historical sketch, to giving prominence only to those periods which are characterized by some advance in our knowledge, or in which some error that obstructed progress was surmounted; and wherever I have left unmentioned any work that may have been serviceable in this direction I trust that the omission will be ascribed to my ignorance of it, but to no other motive.

Among the diseases that were originally grouped together under the title of Bright's disease, my clinical experience and my pathologico-anatomical investigations have led me to distinguish the following essentially distinct processes:

---

[1] Sammlung klinischer Vorträge. Herausgegeben von *Richard Volkmann.* No. 35.

1. *Hyperæmia of the kidneys*—
    a. The active.
    b. The passive, due to venous stasis.
2. *Ischæmia of the kidneys and its results* (the renal affection of cholera).
3. *Parenchymatous inflammation of the kidneys*—
    a. The acute.
    b. The chronic.
4. *Interstitial inflammation or connective-tissue induration of the kidney* (genuine contraction, cirrhosis, sclerosis, granular atrophy).
5. *Amyloid degeneration of the kidneys.*

Now, I am well aware that this classification will not suffice to include all the forms of diffuse changes that are met with in the kidney. There are cases, for example, in which these forms are combined with one another in a variety of ways; but it would serve no practical purpose to establish separate divisions for all these mixed forms, since, from the great number of combinations of pathological states that are encountered and from the varying degrees of these changes, we should be unable to exhaust the anatomical varieties which it would be possible to establish. And, besides this, the divisions could be of no real utility, since with our present knowledge it would probably be impossible to distinguish them at the bedside.

All that I have to say upon these mixed forms, then, I shall incorporate into the different sections into which this work is divided.

1. Hyperæmia of the Kidneys.

a. Active or Acute Hyperæmia.

When active hyperæmia of the kidney occurs, apart from being the anatomical commencement or part symptom of parenchymatous inflammation, we find it is a condition which arises solely as the result of some toxic influence. If the irritating substance that excited it is forthwith removed from the body, the normal state of things is established again in a very short time; and in such cases the active congestion can only be anatomically revealed when the poison has led to the death of the individual by its action upon other organs or upon the nervous system. In some instances, however, the protracted ingestion of some deleterious matter leads to a prolongation of the renal hyperæmia, and this may induce actual inflammation of the kidney. Still, acute nephritis provoked in this way by some special poison is certainly an exceptional occurrence in practice.

I do not enumerate here, among the hyperæmic states, as is often done, those renal processes which are the result of febrile hyperpyrexia and which lead to temporary albuminuria, since the post-mortem examination of these fever patients discovers for the most part no hyperæmia of the kidneys, and because those other cases, where the albuminuria persists to the end of life and the kidneys are found hyperæmic, exhibit distinct evidence of inflammatory swelling and infiltration of the tissues of the kidney as well. I have already laid down my views upon the nature of febrile albuminuria in a previous chapter on Albuminuria, and will only remark here that all other symptoms, so far as the kidneys are concerned, are altogether absent in it.

I may further add that I do not reckon here that condition of hyperæmia of the kidneys which usually follows extensive burns, for, if only the patient survives these considerable destructions of the body long enough, inflammation of the kidneys takes place, as a rule.

Cantharides is about the best known of those poisonous substances, which, after being absorbed into the system, finally provoke hyperæmia of the kidneys; and, whether it be administered internally or applied externally in the form of blister, or as an ointment to keep up a discharge, acute symptoms of renal hyperæmia have been observed to follow its employment.

Very large mustard-plasters and cardol, when these have been applied to an extensive surface of skin, are known to exert an action upon the kidneys altogether similar to that produced by cantharides. In all these cases the effect is no doubt produced by the irritant principle, whether of Spanish fly, of ethereal oil of mustard seed, or of cardol, being actually carried to the kidneys in combination with the blood. Besides the oil of mustard, the oil of turpentine is the only other volatile oil which is of any practical importance in regard to the production of renal hyperæmia; this it produces both when absorbed through the digestive canal and when inhaled in the form of vapor, if only enough of it gain access into the system. Nitrate of potash in large doses also produces hyperæmia of the kidneys.

I do not reckon here the effects produced by certain other poisons, which certainly excite renal hyperæmia, because, as it appears to me, they lead to other structural changes of the kidney tissues besides and beyond mere congestion; to this latter category belong phosphorus, arsenic, sulphuric and other mineral acids, as well as tartrated antimony.

The *anatomical changes* produced in the kidneys by cantharides, oil of mustard, and oil of turpentine are exceedingly little known, since but few opportunities are offered of studying them in the dead body. The only thing that we can safely take for granted is, that both kidneys will be equally affected. (It is self-evident that we should not include under this head of hyperæmia those cases in which actual inflammation of the kidneys has been produced by prolonged ingestion of some one of the irritants alluded to above.) Nothnagel states that after fatal cantharides poisoning "the kidneys present the well-marked aspect of acute renal catarrh, but not that of nephritis." At the post-mortem examination, however, in these cases of poisoning with cantharides, the most characteristic lesion found is the frequently excessive cystitis, a circumstance which has led many writers to believe that the kidneys themselves were not principally affected by this poison. After death by cantharides poisoning, the urinary organs are not the only ones which show signs of inflammation; there is always evidence of inflammatory action throughout the digestive apparatus.

The *symptoms* of renal hyperæmia provoked by the causes above named are invariably accompanied by the concomitant effects which these substances provoke in other organs. Cantharides, taken internally, invariably provoke decided irritation of the digestive organs. Applied externally, they induce, as do also mustard oil and cardol, inflammation of the skin, and at times febrile disturbance as a result of this inflammation. Whether the renal hyperæmia excited by mustard oil and by cardol, like that provoked by cantharides, is at the same time associated with cystitis, I am not able to say. Oil of turpentine absorbed into the system in any quantity, by any channel, ordinarily first gives rise to nausea and vomiting, then sometimes to diarrhœa, and often leads to a state of stupefaction that may proceed up to complete insensibility.

The urinary symptoms provoked in all these instances set in only after a certain period of time has elapsed, and consist at first in a constant and urgent desire to pass water, although no notable increase in the quantity passed usually takes place. In fact, cases have even been reported where, after cantharides poisoning, complete anuria or suppression of urine has been observed; but in such cases it is more likely that true nephritis rather than mere renal hyperæmia was produced. The urine excreted is in many instances found to be very bloody, and in others it contains a few red blood-cells, besides albumen in greater or less quantity; it generally has a few casts in it, but certainly does not invariably present epithelium derived from the tubuli uriniferi. There was no epithelium, for instance, in the urine of a case of mine which owed its origin to the inhalation of turpentine vapor; the same is also true of a case recorded by Dr. Johnson, where a person swallowed half an ounce of oil of turpentine. In cantharides poisoning it occasionally happens that the urine contains such an enormous quantity of fibrin that, if it happen to coagulate within the bladder—as has actually taken place—the resulting clot is large enough to prevent urination. Two of my patients failed to experience any pain in the region of the kidneys; but a third, whose renal affection was entirely provoked by the inhalation of turpentine vapor, complained of pretty acute pain in this situation. He experienced, too, at the same time a painful dragging at the glans penis. Other observers, too, give renal pain as a symptom of acute hyperæmia. According to my own observations, the urine ceased to contain albumen directly the patients were withdrawn from the influence of the pernicious substance which had provoked the trouble. In a sailor in whom the symptoms were provoked by the inhalation of turpentine vapor, the albuminuria only lasted for three days —indeed it had disappeared before the well-known smell of violets had quite left the urine. In the case related by Johnson the albuminuria and the elimination of blood with the urine continued for ten days at least. So in my own two cases of renal hyperæmia provoked by cantharides, the albuminuria and all the other urinary symptoms subsided within a few days. Basham,[1] on the other hand, states that, in contradistinction to the effects of turpentine, the serious consequences entailed by the action of cantharides upon the kidneys do not disappear with the removal of the injurious influence. The urine in one case, three weeks after the patient had ceased to take the tincture of cantharides, still continued to contain free blood-cells and blood-casts.

When the above-mentioned poisons prove fatal, the actual cause of death always seems to be the disorder in its function of some other organ than the kidney.

Mere hyperæmia of the kidneys could not produce a fatal issue, although, as I have already stated, its persistence may induce permanent alterations in the tissues of the organ, and thus an ultimately fatal result.

There is no difficulty attending the *diagnosis* of acute renal hyperæmia directly the cause that has provoked it is recognized, and the symptoms of irritation of the urinary channels which we have described set in; for these symptoms lead to an examination of the urine, which is then found to contain albumen and a varying amount of blood. The spontaneous excretion of fibrinous coagula is especially characteristic of the action of cantharides, and these coagula, as we have stated, may attain such a size as to prevent the escape of urine from the bladder; or, in other cases, coagula of considerable size may form in the vessel containing the urine. It was by such an occurrence as this that I once discovered that an ointment containing cantharides had been clandestinely employed.

---

[1] **Renal Diseases: a Clinical Guide to their Diagnosis and Treatment. London, 1870. p. 12.**

Case IX.—A lady patient of mine, who was in the habit of spending the summer months in the country, had asthma, which troubled her chiefly in close, oppressive weather. She had been for nearly two months at her country-seat, when she drove up to town one day to call upon me, complaining that for some little time she had had considerable difficulty about her water, being tormented with a constant desire to relieve herself, and experiencing also a certain amount of pain. Several times, too, it had happened to her that the urine would not flow at once when she wished it, but, as she herself expressed it, would only start after she had strained considerably, and had succeeded in forcing out some jelly-like masses from the urethral passage. She had emptied her bladder shortly before my visit, and showed me the chamber utensil, at the bottom of which indeed lay a gelatinous, quite transparent mass, surrounded by a considerable quantity of clear but rather deep, yellow-colored urine. This jelly-like mass was of about the size of half the palm of one's hand, and its substance was nearly of the consistence of raw albumen; it contained a few red blood-cells entangled in it. This condition of things at once recalled to my mind an incident which happened to me some time before. I put a blister of ordinary size, that of the palm of one's hand, over the region of the heart, upon a young butcher who had pericarditis in the course of an attack of acute rheumatism. The blister, which had been allowed to remain on for twelve hours, was removed in the morning, and the self-same evening I was sent for, because my patient could not pass his water. I found his bladder fully distended, and passed a catheter for him, through which, first of all, escaped a gelatinous, quite transparent material of the consistency of raw albumen; completely fluid urine subsequently following this. This urine contained a great quantity of albumen, and after a while considerably more fibrin separated itself from the fluid.

To revert: my lady patient denied having employed a blister; but in the course of my examination and palpation of her abdomen, a peculiar odor became apparent, and, upon my further questioning her, she admitted that she had been induced by her doctor in the country to establish an issue on the sternum for the relief of her asthma, an issue maintained by the action of an ointment of cantharides. When she had left off the use of this mischievous material, her urinary difficulties passed over in a few days, and the albuminuria subsided equally rapidly; unfortunately I omitted to make any microscopic examination of the urine of this case.

Case X.—On the 2d of October, 1854, early in the morning, a sailor was brought into the hospital, in an almost insensible state, landed from a ship which had come into our port during the previous night.

The man, who had been previously quite well, had slept in a cabin in which a bottle full of oil of turpentine had broken, allowing its contents to escape in great measure over the floor; the patient's breath smelt strongly of turpentine; not so the contents of the stomach, which he rejected by frequent vomitings. He complained of severe headache; the urine, passed shortly after his admission, possessed the characteristic violet odor, but was clear and contained no albumen.

October 3d.—General condition only a trifle better; head hot; face swelled; pulse one hundred and twelve; complete absence of appetite. The urine, which was passed in moderate quantity, was cloudy, and contained a good deal of albumen—the sediment exhibiting red blood-cells, blood and hyaline fibrinous casts, but no epithelium derived from the renal tubules. The patient complained of a painful sensation of tightness in the region of the kidneys—not increased by pressure, however—and of pain which was directed downwards to the glans penis.

October 4th.—During the night patient had spontaneous bloody and watery stools; but he feels better to-day; face pale; headache has subsided. Patient is again in the full possession of his senses; appetite still indifferent. He complains of a constant desire to urinate and of cutting pain in making water. Urine clear; the sediment contains a number of red blood-cells and casts, of the same character as those observed yesterday; besides which there were some large blood-clots, which could be seen with the naked eye. There was no epithelium from the tubuli uriniferi; the renal pain continued.

October 5th.—Feels completely well; pulse sixty; only slight cutting pain in passing water; urine still smells strongly of turpentine, but contains traces only of albumen.

On the 7th of October urine was free from albumen, and there were no casts in the sediment.

The *treatment* of simple hyperæmia of the kidneys—apart from any complication involving other organs, and produced by one and the same poisonous influence—is confined to the removal of or abstention from the noxious agent, and in any case will consist in prescribing such an abundance of fluids as to procure the rapid washing out of the kidneys. Basham recommends a more active course, consisting of mild oleaginous purgatives, with opium, warm baths, complete rest in bed, mucilaginous drinks, milk and water, and a mild vegetable diet.[1]

---

[1] TRANSLATOR'S NOTE.—The author here makes no direct reference to the employment of camphor, which the translator usually finds noticed as an important and useful remedy for the relief of the hyperæmia produced by cantharides. Several cases of congestive hyperæmia, produced by the injudicious employment of cantharides externally, have come under the translator's care, and he has seldom failed to afford immediate relief by the administration of camphor in doses of from two to five grains every three or four hours.

b. The Passive or Venous Congestive Hyperæmia of the Kidneys

is, as the very term used to designate this condition would indicate, never a spontaneous malady, but always the result of some serious disturbance of the circulation. It may be fairly questioned if any description of this condition ought properly to be made here, since the state of the kidney, as well as the disturbance of its functions, is properly merely a symptom of the fundamental disease. But as views are entertained upon this subject which, it appears to me, are far from clear, I thought it would be better to include the congestive states of the kidney as being within the province of my work.

One may conveniently distinguish two distinct processes, both of which bring about venous congestion of the kidney, although disturbing the functions of these organs in very different ways. For example, there is, first, that general stasis of the venous circulation throughout the body which is produced by valvular lesions of the heart and by certain affections of the lungs; and, secondly, there is a localized venous or partial venous congestion which follows upon obstruction or compression of the vena cava ascendens above the entrance of the renal veins, or even upon the obstruction of these veins themselves.

The plugging of the renal veins, although a pathological event which is often enough observed, happens certainly, as a rule, only upon one side, and then is practically of little or no importance: first, because it occurs most usually when the kidney is otherwise gravely diseased, as by cancer or by amyloid degeneration; and, secondly, because, if involving a previously sound organ, it does so only at the termination of some other process of disease, and constitutes merely a symptom of the agony. Thus I have seen thrombosis of the renal vein in carcinoma of the stomach, and also in the body of a young man, remarkably healthy up to the date of his illness, whose entire left lung was solidified with pneumonic infiltration just then passing into the stage of gray hepatization. For the reasons above given, then, thrombosis of a renal vein produces no such remarkable train of symptoms as to attract special attention, apart from already existing evidence of serious kidney affection, or of the implication of some important organ of the body with grave disease. So far as I know, thrombosis of a renal vein has never yet been diagnosed during life; at all events, if any one has ever ventured to make such diagnosis, he has done so upon insufficient evidence, or upon symptoms that could not positively exclude some other equally reasonable interpretation.

Apart from the other symptoms entailed by the imperviousness of the vena cava ascendens, the effects produced by its being blocked up above the entry of the renal veins are notably different from those which are observed in consequence of congestive hyperæmia of the urinary glands provoked by general stasis throughout the venous system, as witnessed in the course of heart or lung affections.

This difference in the effect produced by renal venous hyperæmia, according to the difference in site of the producing cause—that is, of the obstruction in the course of the circulation—may be explained in the following manner: *where there is obstruction of the lesser or pulmonary circulation, general arterial anæmia must of necessity ensue, and therefore notable diminution take place in the blood pressure upon the secreting vessels of the kidneys. A like result, however, is not brought about by obstruction of the ascending vena cava.* The venous blood collected from the lower half of the body, although diverted from its natural channel towards the right heart, reaches this at last by collateral branches; and while at first this collateral venous circulation may not fully suffice, the arteries, nevertheless, may remain sufficiently full throughout the entire time, owing to the fact that the flow of blood from the superior cava and the hepatic veins remains undisturbed. For this very reason, in a case of obstruction of the vena cava inferior, the degree of pressure in the branches supplying this vein will rapidly become excessive, and will necessarily give rise to an œdematous swelling of the lower half of the body. If we picture to ourselves the effect which this obstruction must have upon the conditions of pressure in the renal vessels, it will at once be apparent that the first result must be a considerable over-filling of the renal veins and an increase of blood-tension within them; and although anastomosis of the renal veins with those of the cellular tissue surrounding the kidneys admits the development of a collateral circulation (by the intermediation of the capsular, and the collecting intra-renal veins on the one side, and the vena azygos and hemi-azygos on the other, which open into the superior vena cava), the blood pressure within the renal veins must still remain excessive. At the same time the renal arteries will continue to be filled nearly up to the same pressure point that prevailed before the obstruction took place. The inevitable result of this is a preternatural increase of blood pressure, which makes itself felt especially in the glomeruli, and which leads not only to increased secretion of urine, but also to the escape of albumen and probably, through bursting of some of the capillary coils within the glomeruli, to the passage of considerable quantities of blood into the urine. Cases of thrombosis of the inferior cava, in persons otherwise moderately healthy, are very rare; I have not been able to find a single case recorded in which sufficient attention appears to have been paid to the manner in which the renal functions are fulfilled. I communicated a case of this kind early in this work (Case I., p. 45).

Far more frequent in practice, however, are the cases of general venous stasis. It is to these only, therefore, that we shall have reference in the following remarks. With this condition, diminished fullness and tension of the aortic system is inseparably associated. Its effect, therefore, upon the functioning activity of the kidney must be totally different from that produced by local disturbance of the circulation through the vena cava inferior.

Etiology.

Only the higher grades of general venous stasis exert any appreciable effect upon the kidneys and their functions. The higher grades of general venous congestion are most commonly produced by heart diseases; and among these last we are chiefly concerned with the valvular lesions, so soon as the natural modes of compensation cease to obviate the disturbances which these produce in the circulation. Foremost in frequency belong here the obstructive valvular lesions at the mitral orifice; but those cardiac affections, which are characterized by some morbid change in the muscular structure of the heart, may exert an equally disturbing influence upon the distribution of the blood, provided the degeneration of the heart's muscular structure has reached such a degree that this central organ is inadequate to its task of carrying on the circulation. For example, I have observed a high degree of general cyanosis and severe functional disturbances of the kidneys following upon atrophy of the heart muscle, which in turn had been produced by an indurative pericarditis. Moderate pericardial exudation I have never seen followed by a similar amount of disturbance of the general circulation.

In respect to pulmonary affections, as causes of congestive hyperæmia generally, and of renal congestion in particular, we may remark that the effect thus produced is by no means, as some have imagined, directly proportionate to the obliteration of branches induced in the lesser or pulmonary circulation. The commonest forms of lung disease that lead to obliteration and destruction of extensive ramifications of the pulmonary artery, *i. e.*, the pathological processes connected with phthisis, very rarely give rise to symptoms of general venous stasis; the reason for this being, as above remarked, that the total bulk of the blood is so quickly lessened by the accompanying hectic fever that the pulmonary vessels which have escaped destruction offer room enough for the transmission of the blood that remains. Those lung diseases which, like emphysema and certain forms of interstitial pneumonia, lead to extensive obliteration of capillaries in the pulmonary parenchyma, and sometimes produce diminution in the calibre of the larger branches, or, more frequently, narrowing and obliteration of numerous ramifications of the pulmonary artery, are the ones that produce the gravest states of general venous stasis, and sometimes venous hyperæmia of the kidneys and the symptoms which mark this. On the whole, however, this state of venous stasis and functional disorders of the kidneys are far less commonly encountered in pulmonary than in heart diseases, and, besides, in the former they are far less likely to lead to serious results.

I have never known either grave venous stasis in the kidneys, or any serious disturbance of the renal functions, to occur as a result of pleuritic exudations or of pneumothorax.

Pathological Anatomy.

In my account of the pathological conditions found in this form of disease, I shall confine myself to that state of the kidneys recently described under the title of *Cyanotic Induration of the Kidneys.* Klebs,[1] gives the following description of it: "The kidneys are larger than they should be, and are surrounded by a capsule which is provided with very little fat; the capsule proper can be stripped off easily, and the surface of the organs looks vascular, but perfectly smooth—the venous radicles, or star-like commencements of the veins, appearing enlarged and filled to distention. The whole organ is considerably firmer than it should be, and does not diminish in firmness after the blood has drained off from it. Upon section, both medullary and cortical substances are seen to be highly vascular, although the medullary cones are the more deeply colored of the two, by reason of the specially marked engorgement of the vasa recta. In the cortical substance the vascularity is generally diffused throughout the capillaries, the glomeruli not appearing overdistended. Microscopical sections show the hyper-distention of the veins and capillaries extending back even to the Malpighian tufts. The epithelium of the tubuli is ordinarily unaltered; on the other hand, the interstitial tissue is unwontedly tough, but not broader, or only very little broader, than normal; even in the fresh state, it admits of easy demonstration by brushing out the cells, and displays its fibrous texture more clearly than in the normal condition.

"This change may exist for a long while without causing any disturbance of the renal functions, although a very little increase of the arterial tension may suffice to allow albumen or blood to escape from the glomeruli, owing to the fact that the outflow of the venous blood takes place with difficulty. Another danger, however, exists, that, namely, of secondary disease of the kidney provoked by its mal-nutrition, for the blood thus stagnating in its capillaries is poor in oxygen and overloaded with carbonic acid, and does not provide proper nutrition for the organ; under these circumstances a granular degeneration of the epithelial cells takes place, chiefly affecting those which line the curling cortical tubes; the cortical substance appears then of a pale grayish-red color, and stands out in marked contrast with the deeply cyanotic medullary substance. This peculiar color of the cortical substance is due to the fact that the swelling of the epithelium in the cortical portions squeezes the blood out of the capillaries surrounding them, and then, on section, the glomeruli —from which the outflow per vasa efferentia has been impeded— stand out against the surrounding pale parts as dark-red points, and not unfrequently some of their capillaries burst in consequence of this obstruction to the outflow, and discharge their blood into the tubuli uriniferi."

---

[1] Handbuch der pathologischen Anatomie. 3. Lieferung, S. 631. Berlin, 1870.

Klebs describes a further danger, namely, the possible occurrence of interstitial changes, which take place chiefly in the superficial cortical portions. Thus in recent cases one may find the interstitial connective tissue around the glomeruli and the commencements of the urine tubes beset by lymphatic elements; and later on, perhaps, cicatricial connective tissue may form here, and through its subsequent contraction cause the obliteration of a number of urine tubes and Malpighian bodies.

Rindfleisch[1] disputes the occurrence of a congestive nephritis, but admits a proliferation of the interstitial connective tissue, —regarding this, however, as the result of its hyper-nutrition, since it bears all the characters of being an homologous development.

While Bright pronounced the changes induced in the kidneys by general venous stasis to be something different from kidney disease proper, and while Traube after him maintained that they should not be looked upon as inflammatory in origin, Lecorché alone has quite recently described them as constituting a peculiar form of his interstitial nephritis or sclerosis.

It certainly admits of dispute, however, whether the increase of interstitial connective tissue—a process brought about in so many organs of the human body by persistent venous congestion, and by which the implicated organs become so remarkably tough—should be regarded as inflammatory, or not. According to the ordinary acceptation of the term, it certainly should not be so regarded. The brown pigment induration of the lungs, observed in connection with valvular defects at the mitral orifice, is not designated as an interstitial pneumonia; and the thickening of the peritoneum, that follows upon portal congestion, no one thinks of calling peritonitis. In the congested kidney, at all events in every pure example of it, the characteristic accumulation of lymphoid elements in the interstices between the tubuli is not discoverable, and I have never seen inflammatory swelling of the epithelial cells in the tubes, though I will not dispute the occurrence of a fatty degeneration, that is, of an atrophic wasting, of these cells. I attribute the shallow, cicatricial depressions —such as I have often seen in large numbers upon the surface of the kidney in old-standing cardiac cases—to the atrophy or wasting of a certain number of curling tubes, together with the glomeruli belonging to them; but these are of course to be distinguished from the deeper, more penetrating scars that mark past embolic processes, such as are so often found among the results of long-continued renal venous hyperæmia. I have never, however, witnessed any contraction of the kidney to a size below normal as the consequence of persistent venous congestion, but I have often seen cases of genuine contracted kidney in which the patient, while still under my observation, has had endocarditis, affecting the mitral valve and leading to its permanent defectiveness; and from this I am led to think that, in the cases of contraction of the kidneys supposed to be due to persistent venous congestion in consequence of heart disease, confusion has arisen regarding the actual sequence of events in point of time.

---

[1] **Lehrbuch der pathologischen Gewebelehre. Leipzig, 1867–69, S. 436.**

Rosenstein,[1] it should be stated, offers another hypothesis in explanation of this combination of atrophied kidneys and cardiac valvular lesions; he regards both events as co-results of one and the same fundamental malady—rheumatism. But to this I shall revert later on, in my account of acute nephritis.

---

[1] l. c. S. 54.

Among the changes found in the body after death, besides cyanotic induration of the kidneys and the causes leading to it, I must yet direct special attention to the dropsy: first, because it is an almost invariable coincident symptom; and, next, because it lies in close causal relationship to the functional disorders of the kidney associated with general venous stasis. In nearly every case of this kind the dropsy is confined to that part of the body which is drained by the vena cava inferior, although, to be sure, one often finds considerable effusions in the serous sacs of the thoracic cavity—the head and arms being, as a rule, free from dropsical swelling. This peculiar distribution of the dropsical effusions, differing as it does so entirely from that which occurs in the course of kidney diseases proper, is due to the unequal manner in which the blood is distributed throughout the veins, and is connected with the disturbance in the renal functions only in so far as this predisposes in a general way to dropsical effusions. The mechanical reasons for this so constant limitation of the dropsy to the regions drained by the vena cava inferior, in all heart diseases, and especially in valvular affections of the left ostium venosum, I have already endeavored to explain elsewhere.[1]

Symptoms.

In my description of the symptoms associated with congestive (venous) hyperæmia of the kidneys, I shall take into consideration those produced by the fundamental malady (whether heart or lung affection) only in so far as they assist us in understanding the functional disorders of the kidneys. These symptoms consist, on the one hand, in a general overfilling of the veins, advancing even to well-established cyanosis, and, on the other, in diminished fullness throughout the entire arterial system—an emptiness sufficient to render the radial pulse small and feeble, and in extreme instances even imperceptible; in short, the symptoms are those of extreme elevation in the tension throughout the venous system, with a corresponding diminution of tension throughout the arterial system. Disturbances in the rhythm of the movements of the heart and other irregularities of the pulse do occur; but they are not constant, and, besides, they are not symptoms which interest us in this connection.

---

[1] Deutsches Archiv für klinische Medicin. Bd. 4, S. 269. Anmerkung.

If we now take the renal symptoms provoked by general venous stasis—as induced principally by valvular lesions of the heart, insufficiently compensated—and compare them with those caused by partial venous stasis, such as results from obstruction of the vena cava, we shall perceive that the disordered functions of the kidneys, in cardiac deficiency, are only partially due to venous hyperæmia of these secreting organs; they are, in fact, largely due to the associated diminution of arterial pressure.

It is ordinarily not until the heart defect has existed for some time, and cyanosis of the face has indicated the accumulation of venous blood in the peripheral veins and the inadequateness of the compensatory arrangements to overcome the obstruction to the circulation, nor until breathlessness upon every slight exertion has grievously impaired the facility of locomotion of the affected individual, that dropsy, beginning by anasarca of the lower extremities, directs attention to the renal functions. In every such case one finds that the quantity of urinary water excreted daily falls far below the normal average. This scanty urine, too, is usually of a dark, brownish-red color, and, although clear when first passed, quickly becomes thick, through an abundant deposition of urates; its ordinary reaction, when there is no special influence at work to affect this, is strongly acid. This acidity of the urine in heart disease appears often to be due to the presence of a free acid, since uric acid is often excreted with the urine, either in crystalline form or in the shape of small concretions (gravel). The specific gravity of the urine is always raised above normal, reaching to 1030 or even beyond 1035. The urine, as might be inferred from this high specific gravity, contains a large quantity of solid constituents, among which the urea, by reason of its quantity, occupies the first place. The percentage content of urea in such urine as this may even exceed five per cent. The urine in heart disease, too, as a rule, is much richer in uric acid than that furnished by healthy individuals under ordinary conditions, both in percentage and in absolute quantity.

Directly dropsy has begun, one finds the urine also constantly albuminous. The quantity of albumen remains, however, as a rule, small, seldom reaching to two parts per thousand. In such cases we find that, coincidently with the appearance of albumen in the urine, the sediment examined under the microscope presents small, pale, homogeneous casts, although never in large quantity ; rarely a few scattered epithelial cells out of the tubuli stick to them. Besides casts we very often find in the sediment some scattered red blood-cells, but never so many as to give the urine a red color in consequence. Should this be the case, a hemorrhagic infarctus produced by embolism may be conjectured to have taken place in addition to the existing venous hyperæmia.

There is no pain or even any decided tenderness on pressure in the region of the kidneys in venous hyperæmia ; but should these symptoms be present, they would indicate that, in addition to the hyperæmia, a hemorrhagic infarctus had formed or that calculi were present in the pelvis of the kidney, both of which complications I have seen. The swelling of the kidneys, under these circumstances, takes place too gradually to produce any considerable irritation of the nerves distributed to the capsules of these organs.

As above remarked, the attention of the medical man is, as a rule, directed to the renal functions of a patient suffering with heart disease, only after dropsy has set in, and then he discovers just that character of the urinary secretion which I have previously described. The quantity of fluid secreted remains scanty so long as the dropsy is on the increase, and all this time the urine retains the features above mentioned, being of high specific gravity, albuminous, etc.

When once established, these disturbances of the renal functions, in persons suffering from heart disease, may persist, without any remission, up to the end of life, and death is then usually ushered in by continually advancing dropsy and increasing difficulty of breathing—provided no process of embolism or other accidental complication, derived more immediately from the heart, serves to extinguish life more suddenly. So far as my own experience extends, *uræmic symptoms are never associated with heart disease, unless some other complication beyond mere cyanotic induration co-exist*, and this fact Traube has already insisted on.

In other examples of heart disease we see the functional disturbances of the kidneys subside again completely, and this, too, often quite suddenly, so soon as the patient is removed from unfavorable conditions and is placed under such as favor his recovery; as, for instance, when miserable, neglected creatures of the vagabond class come, as I have so frequently had the opportunity of observing, to enjoy the comforts of a good hospital. As is well known, in neglected cases of heart disease wonderfully rapid improvement of apparently the most hopeless conditions is often effected by medical treatment when the degeneration of the heart's muscular substance is not yet too far advanced.

In other instances, one perceives a fluctuating energy of the kidneys, lasting for considerable periods. Thus for months the daily quantity of urine secreted may be scanty, the fluid presenting a high specific gravity and containing albumen; the dropsy too persists obstinately all the while. But yet even then an abundant secretion of urine may be re-established, the albumen disappear, and the dropsy too entirely subside. I have known patients with heart disease, who had been unable to leave their beds for half the winter, to enjoy afterwards years of relative health, before permanent cardiac insufficiency at length provoked dropsy afresh, and finally led to death.

Analysis of the Symptoms.

That the disturbances of the renal functions in heart disease do not depend upon the anatomical state of the kidney—the so-called cyanotic induration—but simply and entirely upon the impairment of the equable distribution of the blood, caused by the cardiac lesion, we are taught in the most incontrovertible manner by a careful and prolonged observation of the symptoms that take place in the vascular system, and of the bearing that these have upon the activity of the kidneys. The same kidneys, which for months had secreted small quantities of dark albuminous urine of high specific gravity, so long as the tension in the general arterial system remained far below normal and the cyanotic color of the patient demonstrated the serious over-repletion of his veins, secrete ample quantities of urine free from albumen, clear, and of normal specific gravity, so soon as, in consequence of some spontaneous process of cure, or by interposition of medical skill, the unequal distribution of the blood is once more equalized, the natural blood pressure being restored to the arterial system, and the abnormal increase of tension in the veins becoming subdued. The following case will serve as proof of this.

CASE XI.—J. L., a field laborer, aged twenty-two years, born in Posen, was admitted November 24, 1874. Patient states that he has never been really ill before, and that he felt perfectly well up to two months ago. At that time, however, upon exerting himself unduly or going about rapidly, he experienced some shortness of breath, and slight anasarca of his feet manifested itself. This swelling, however, soon increased to such a degree that for the last three weeks he has been quite unable to continue his employment.

Condition on Admission.—Is a slightly built man, of scarcely middle height, having feebly developed muscles, and but little fat upon him; face cyanotic; ankles and feet highly dropsical, and the skin over them somewhat purple-colored; thighs less swollen, but considerable œdema of scrotum and abdominal walls exists, as well as of the subcutaneous connective tissue at lower part of back. Ascites moderate.

Thorax.—Cartilages of ribs on left side towards lower part of sternum are bulged considerably forwards (*voussure*); the heart's impulse is visible and can be felt on the left side of the sternum in the fourth, fifth, and sixth intercostal spaces,—in the sixth space to a distance of four ctm. outside the left nipple. At the left margin of the ensiform cartilage, in the pit of the stomach (anticardium), there is a distinctly visible and palpable systolic impulse. Well-marked undulations are visible in the jugular veins of the neck. No liver pulse could be detected with certainty. The cardiac dullness extended beyond the normal limits in every direction: upwards it extended to the second left intercostal space, upon the left side; towards the left, it extended a distance of four ctm. outside the mammary line; and towards the right, three ctm. beyond the right border of the sternum. The liver dullness extended downwards on the right side in the mammary line, to three ctm. below the right costal arch.

Auscultation discovered a systolic, and besides this an exceedingly rough diastolic murmur at the apex-beat of the heart. A systolic murmur, whose character differed essentially from that heard at the apex of the heart, was distinctly audible at the left margin of the ensiform cartilage, at the point of visible pulsation, in the pit of the stomach. Aortic sounds feeble but clear; accentuation of the diastolic sound over the pulmonary artery; pulse 116, small, feeble, irregular; respirations, 28; urine dark colored, and estimated to contain about 0.2 per cent. of albumen.

Diagnosis.—Insufficiency with stenosis of mitral valve; dilatation and hypertrophy of right ventricle; relative insufficiency of the tricuspid valve; passive hyperæmia of liver and kidneys; secondary dropsy.

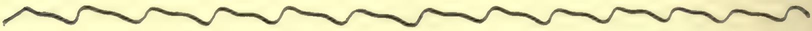
November 26th.—Ordered infusion of digitalis and neutral mixture, equal parts: a tablespoonful to be taken every two hours; complete rest in bed. Under this plan of treatment the patient improved very rapidly; the pulse diminished in frequency, sinking first to 84, and then to 72; it became regular and full, and the arteries felt tense; the cyanotic color of the face and extremities disappeared, the urine increased in quantity. On the 30th November there was scarcely a trace of albumen to be discovered in the urine, and the dropsy had disappeared.

The following table gives a summary of the daily secretion of urine during this period.

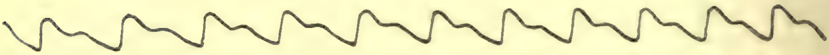
Date.	Quantity of Urine, in cub. cent.	Spec. Grav.	Albumen.		Urea.		Remarks.
			Per Cent.	In Toto.	Per Ct.	In Toto.	
Nov. 25...	542	1026	0.024	0.13	5.0	27.1	On the 24th of November the albumen amounted to 0.2 per cent.
" 26...	510	1030.5	a trace				
" 27...	890	1032	a trace				
" 28...	620	1024	a trace				
" 29...	3580	1010	a trace in day's urine.				
" 30...	4160	1010	0				
Dec. 1...	2900	1007.5	0				
" 2...	2870	1009	0				
" 3...	2340	1013	0				
" 4...	1710	1015	0				

On December 2d the digitalis was left off; from this time on the quantity of urine secreted began to diminish, and fell, in the course of the next few weeks, to about 850 c. ctm. per day, remaining at this low figure for several days. Traces, too, of albumen were again found in the urine, notably in that which was passed during the day-time, when the patient was out of bed; but with the re-establishment of the patient's strength, the urinary secretion increased again, without the employment of any medicine, and the albumen once more disappeared from it.

Now there can be no doubt that in this and other similar cases the diminution in quantity of the urine is simply attributable to the diminution of the arterial tension. For, upon the restoration of the natural pressure throughout the arterial system,[1] there at once ensues an abundant secretion of urine, and this increase may even be so great that—both in the present case and in others where I have given attention to this point—the actual quantity of fluid drained off by the kidneys may be largely in excess of that which has been taken in drink or otherwise ingested with the food. Upon the same conditions, too, the variation in the specific gravity of the urine depends.



CURVE I.



CURVE II.

In many cases of heart disease the functions of the digestive organs and the assimilation of food may proceed tolerably well, although decided symptoms of cardiac insufficiency have set in; and, in correspondence with this fair fulfilment of the nutritive processes, nearly normal quantities of nitrogenous excreta are furnished by the tissue interchanges, and pass into the blood. In cyanotic induration of the kidney, the epithelium preserves its normal properties, and continues to fulfil its function of separating the products of nitrogenous waste from the blood. The small quantity of urinary water which the glomeruli furnish, when filtering under feeble pressure, leads to the concentration of the secreted fluid or its high saturation with the solid constituents of the urine. In persons who are extremely reduced, and in whom the nutritive interchanges are brought down to a minimum, one finds the urine, although greatly diminished in quantity, often of low specific gravity.

---

[1] The appended sphygmographic tracings show the pulse waves observed in the case of the patient L. Curve I. was taken on the 23d of January, when the secretion was very small and the urine albuminous; Curve II. on the 21st of February, after the patient had taken digitalis, when the secretion was abundant and the urine free from albumen.

Further, the uric acid, among the specific elements of the urine, often occupies, so far as actual quantity is concerned, an important position, reaching, in some cases of heart disease that I have observed, to the proportion of one to twenty in relation to the urea; but this depends not upon the condition of the kidney, but upon the influence which the disturbance of the respiratory functions in heart disease exerts upon the tissue interchanges and the products issuing from these.[1]

The relatively, and often absolutely, large quantity of urates contained in the urine in heart disease is the cause of the abundant and ordinarily intensely colored sediment which forms in it on its cooling. The comparatively small quantity of urine water furnished cannot hold these salts in solution, first, because there is too little of it, and, second, because under the conditions above noticed, the urine, as a rule, presents a strongly acid reaction, and the urate of soda contained in it is therefore in the form of an acid salt, and one that is with difficulty soluble. I have already above expressed my surmise, that a free acid is likely often to be found in the urine of patients suffering with heart disease; at least, I cannot otherwise explain a symptom which I have so often observed, namely, that the uric acid, in patients suffering from heart disease, separates itself from its combination with its base while still within the urinary channels, and is eliminated in the crystalline form, or while still within these channels forms small concretions (gravel). The catarrhal swelling of the mucous membrane lining the pelvis of the kidney, which is so often present in congestive (passive) hyperæmia of these organs, with the abnormal separation of fermenting elements which it implies, may perhaps exert some decomposing action upon certain of the constituents of the urine, and thus give rise to the formation of a free acid (Meckel and Scherer[2]).

---

[1] Compare *Bartels*, Harnsäureausscheidung in Krankheiten. Deutsches Archiv f. klin. Med. Bd. 1, S. 13.

[2] Compare also *Neubauer* und *Vogel*, Anleitung zur qualitativen und quantitativen Analyse des Harns. 6. Auflage, S. 109. Untersuchungen von *Voit* und *Hofmann*.

I consider the albuminuria of heart disease to be the consequence of the abnormal increase of blood pressure in the renal veins, and assume that the albumen does not pass through the capillaries of the Malpighian tufts, but is forced through the walls of the intertubular capillaries directly into the urine tubes. By the same channels a few red blood-cells emigrate and reach the urinary secretion. The reasons for this have been already stated by me in detail (page 46, et seq.). The albuminuria comes and goes in heart disease with the appearance and disappearance of the cyanosis, with the depression and elevation of the arterial tension.

In uncomplicated cases of passive hyperæmia of the kidneys the quantity of albumen contained in the urine is quite insignificant, seldom reaching to more than 0.1 per cent.; and since the excretion of albumen in this disease only occurs when the urinary secretion has been reduced to a minimum, the actual loss of albumen which the system suffers in this way does not exceed a fraction of a gramme for the entire twenty-four hours. Such a loss as this can exert no influence either upon the nutrition of the body at large or upon the composition of the blood, and therefore can have no direct bearing upon the appearance or subsidence of the dropsy.

On the other hand, the appearance or subsidence of dropsical swellings in heart complaints has the very closest dependence upon the quantity of water that the kidneys excrete. If the elimination of urine falls far below what is normal, by reason of diminution in the arterial pressure, water is retained in the blood, and the serum becomes thin and hydræmic. Then, as a further result, dropsical swellings take place in those parts of the body where the veins are most highly congested with blood, —within the province, therefore, of the inferior vena cava. If, under these circumstances, we should succeed in overcoming the circulatory disturbance and in restoring the normal arterial tension, the kidneys often separate enormous quantities of aqueous urine from the impoverished serum, and the dropsy rapidly disappears.

Course and Terminations.

The progress of the symptoms in passive or venous hyperæmia of the kidneys depends entirely upon the nature of the original malady which gave rise to the general venous stasis. If this is of such a character that the circulatory disturbance which has been produced, admits of diminution or compensation, as when a newly instituted mitral deficiency becomes compensated by hypertrophy of the right ventricle, the symptoms of renal hyperæmia, if any such have manifested themselves, fall into abeyance so long as the compensation is adequate. On the other hand, the renal symptoms become permanent, when the compensatory provisions are no longer sufficient to overcome the obstruction to the circulation, when—to hold fast to the example already offered—the muscular substance of the hypertrophied ventricle has at last become fattily degenerated, and the heart's pump action steadily more and more impaired. The daily quantity of urine secreted then becomes progressively scantier, until at last the secretion may nearly cease altogether before the patient dies; death ensuing then by dropsy of the most extreme kind, if not provoked beforehand by some intercurrent accident, cerebral embolism, pulmonary apoplexy, paralysis of the heart, or the like.

That uræmic accidents do not occur, as already remarked, in the functional renal disorders provoked by venous congestion, is due in part to the circumstance, that in the indurated cyanotic kidney the epithelial lining of the urine tubes remains entire and sound, and that, although the quantity of water separated by the Malpighian tufts is reduced to a minimum, it still suffices to eliminate the excrementitious urinary constituents from the blood and to prevent any accumulation of them either in this or in the tissues. This is verified, too, not only by the clinical observation of patients suffering from heart disease, in whom often very large amounts of the specific urine constituents are found in small quantities of urine, but also by the very ingenious and instructive experiments of Heidenhain and Neisser.[1]

---

[1] Versuche über den Vorgang der Harnabsonderung, in Verbindung mit Herrn Stud. med. *A. Neisser* angestellt, von *R. Heidenhain*. Pflüger's Archiv für die gesammte Physiologie. Bd. 9, S. 1.

In these cases of heart disease the appearances found at the autopsy may often be interpreted as affording evidence against the universal applicability of Traube's explanation of the uræmic manifestations. Grave degrees of brain œdema, such as are for the most part found in the bodies of those who have died of renal disease with uræmic symptoms, are by no means uncommon at the autopsies of persons who suffered from heart disease, but who yet had not shown any symptoms of uræmia. That in heart disease there exists an over-repletion of the veins besides this œdema, cannot invalidate this objection to Traube's theory. Kussmaul and Tenner, to whose experiments Traube refers, perceived epileptiform twitchings in animals whose tracheas only had been ligated; the supply of arterial blood to the brain being thereby, as a matter of course, interrupted, while the venous supply, however, still continued. In determining the question of why uræmic symptoms are not forthcoming in heart disease, notwithstanding the extreme diminution of the urinary excretion, there remains still another factor to be taken into account, namely, that in this last stage of heart disease the tissue interchanges, for evident reasons, are seriously interfered with, and that consequently only insignificant quantities of excretory material are formed, of which quite a considerable quantity may be lodged in the dropsical effusions, and thus remain outside the blood and lymph vessels.

Diagnosis.

Any obstruction offered to the blood current, through the entire venous system, must also, of necessity, implicate the renal veins; and a serious grade of cyanosis enduring for any length of time permits the inference, even without further evidence, that the kidneys have, by reason of this obstruction, suffered the so-called cyanotic induration.

On the other hand, the appearance of albumen in the urine in heart disease does not at all justify the deduction that this abnormity of the renal secretion is the result of a stagnation in the circulation through the veins. Albuminuria in cardiac affections may be caused by hemorrhagic infarctions of the kidney, or it may result from the complication of a previously existing renal disease—granular atrophy, for instance. I have never seen either parenchymatous nephritis or amyloid disease associated with a chronic heart affection which produced general venous stasis.

Besides the evidence of the heart disease, the peculiar features of the urine, its small quantity, high specific gravity, and slight content of albumen, should be sufficient to assure the diagnosis of cyanotic induration of the kidney, or at least of the existence of that permanently abnormal distribution of the blood by which it is produced.

In hemorrhagic infarctions of the kidney the quantity of the urinary secretion need not necessarily be diminished; indeed, a diminution takes place only when the general arterial pressure is lessened and that in the veins is increased. Occasionally when large infarctions form, the patients complain of pain and tightness in one or other renal region. If a large infarction causes albuminuria, one usually finds so much blood in the urine that a visible blood-colored sediment is formed, though this certainly does not occur in every specimen voided. The sediment also contains blood casts, a feature which I have never seen in simple passive congestion of the kidneys.

In the cases I have observed of valvular lesions of the heart, complicated with granular atrophy of the kidneys, no serious grade of venous stasis was ever reached. The urine, therefore, presented the same characteristics which it ordinarily presents in the above-named renal malady: it remained pale, and, in one instance, was of low specific gravity up to the very last, although the daily quantity of urine passed became scanty with the progressive loss of the patient's strength. This case terminated with uræmic convulsions; the cardiac affection had evidently been established long after the existence of the renal disease. It appears to me doubtful if disorders of the circulation, originating in valvular lesions and associated with advanced granular atrophy, can go on to the production of extreme and lasting cyanosis; the patient, it seems to me, would succumb before the cyanosis became developed.

Prognosis.

Venous hyperæmia of the kidneys is always merely a symptom of disease in some other organ, and possesses, so far as the prognosis is concerned, only this value, it shows that functional disorders which accompany this condition of the kidneys constitute an important link in the chain of symptoms, and are proof that the circulation has suffered some serious disturbance. If this embarrassment of the circulation is overcome, such alterations of the renal structures as have been meantime produced, entail no important after-events ; but in the larger number of cases the defects in the circulatory organs are of an irreparable nature, and while the disturbing effects of these can be counteracted for a long time by natural compensatory processes, and for brief periods by the use of certain remedies, they sooner or later lead to permanent inadequacy of the natural blood-driving forces, and consequently to a premature death, which, it is true, is materially hastened by the failure of the kidneys to perform their functions.

The duration of the renal malady depends, self-evidently, upon the nature of the primary disease. Ordinarily the renal disorders in heart disease pursue a chronic course, fluctuating according to the variations in the energy of the heart, but increasing, as time passes on, with the aggravation of the cardiac affection. The renal symptoms may be moderately acute, and may amount to a degree threatening immediate danger, as when some fresh attack of endocarditis occurs in a case of old valvular deficiency. We must not forget, however, the possibility of repair and of a return to relative health.

Treatment.

The treatment of this condition of things turns naturally, in the first place, upon the original malady. If we succeed in overcoming the disturbance in the circulation, the functions of the kidneys will speedily be re-established. But since, in most cases, as stated, there exists an irreparable fundamental malady, our attention must be, as a rule, directed to taking careful observation of the way in which the central organ of the circulation performs its duties; and since it undergoes, as is well known, extraordinary vicissitudes in the course of the chronic affections to which it is liable, we should, if possible, at the very commencement of serious irregularity in its action, take those measures which the immediate condition requires. The violence and rapidity of the heart beats, which so often interfere with the complete filling of the left ventricle in mitral deficiency, may be subdued with certainty by digitalis. Our thanks are due to Niemeyer for having correctly laid down the indications for the employment of this wonderful remedy in the condition above mentioned. Under its use the heart's action becomes quieted in the course of a few days, the normal and equable distribution of the blood becomes restored, the cyanosis disappears, the nearly arrested urinary secretion returns in abundance, and the dropsical effusions soon become absorbed.

In other cases the feebleness of the heart's action may compel us to employ the most powerful stimulants—camphor and musk; for under these conditions stimulants become the best diuretics.

It is not, however, my task to discuss here the entire therapeutics of chronic heart disease, but to treat merely of that state of things which the renal disturbance and its sequelæ specially produce. In this direction, therefore, I shall address what remarks I have to make. We have to consider, then, the abnormal diminution of the urine and the dropsy, which become the object of our special attention, when the therapeutic measures above indicated no longer suffice to regulate the circulation and distribution of the blood, and thus to procure a requisite discharge of urine.

As to specific diuretics, much must not be expected of them, so long as the filtration pressure upon the Malpighian capillary coils continues to be decidedly below the normal standard. At the same time, I am not at all prepared to deny to diuretics a beneficial action. In particular, I fancy that the potash salts combined with an organic acid may, even under the above conditions, exert a diuretic effect. Their employment has the additional effect of neutralizing the acidity of the urine, since the potash compounds, by becoming further oxidized within the blood, are eliminated as carbonate of potash from the system (through the kidneys). Thus the risk of formation of concretions from the crystallizing out of uric acid within the renal passages is obviated. In such cases of chronic cardiac affection, with secondary venous hyperæmia of the kidneys, in which digitalis is indicated, I ordinarily prescribe this drug, in combination with a neutral salt of potash, according, for instance, to the following formula: a tablespoonful every two hours, of a mixture of two parts of neutral mixture to five of infusion of digitalis.

In the dropsy of heart disease I have never seen the essential oils or acrid vegetable diuretics exercise like effects in increasing the urinary secretion, and I have therefore abandoned their use.

With the means above given, however, one often does not succeed, in cases of passive hyperæmia of the kidneys, in augmenting the urine and subduing the dropsy. We are compelled in these cases to drain the system of water by other channels, in order to relieve the discomfort and remove the danger with which the increasing dropsy threatens the patient; and, to this end, we may resort to derivation, either by way of the skin or by way of the intestines. The old physicians preferred attaining their object by promoting watery diarrhœa—partly, probably, because an efficient diaphoretic treatment is always fraught with greater trouble than the administration of some drastic purgative pill or decoction of colocynth, and partly because the very handy practical modes of procuring diaphoresis now possessed by us were not known to them, but more perhaps because they feared that in the use of energetic sudorific measures they would subject patients suffering from heart disease to undue risk.

According to my experience, gathered in the earlier years of my practice, the drastic purgatives nearly always fell short of their aim of removing cardiac dropsy. The dropsy was not gotten rid of in this way, but the means employed were certain to upset the patient's digestive and assimilating powers very greatly; indeed, it is only necessary to take the trouble of examining the watery fæcal stools passed under these conditions to find peptones in them regularly, and often in abundance, if not, as often happens as well, albuminous substances in solution. Now the result of such proceedings must be the further weakening of an organism already brought quite low enough in its nutrition; and in this general exhaustion the previously damaged heart, too, must participate. It is for this reason that I hold the treatment of cardiac dropsy by drastics to be altogether objectionable.

On the other hand, we ought to resort, without any sense of fear, to the second method of removing water from the system, namely, by promoting the sweat secretion of the skin. With this object in view, no one would nowadays employ the once favorite drugs, spirit of Mindererus, etc., substances reputed to possess a sweat-promoting action. A really efficient diaphoresis, one which is to drain water away from the body, without the administration, either at the same time or just before, of a suitable quantity of some hot or cool drink, can be accomplished only by heating the skin through some agency which acts upon it from without. This application of heat to the surface may be made by maintaining the body in hot air or hot vapor baths, in steam closets, or in warm sand baths, or by surrounding the person with wet wrappings, and then preventing surface evaporation by extra coverings.

The method, however, most agreeable to the patient, and at the same time also, as I believe, most effective, is to heat the skin by dry hot air, as is done in the so-called Turkish bath. This plan is greatly to be preferred to vapor and steam closet baths, since thereby any actual overheating of the body is avoided, although the atmosphere of the sudatorium is commonly raised to above 50° R. (145° Fahr.). The sweat, which pours freely from the whole surface of the body, when such a temperature as this is reached in the chamber, rapidly evaporates in the dry hot air, and the heat required to convert the palpable moisture (standing on the body in the form of drops) into vapor is extracted in part from the surface of the body, and in part from the layer of air which lies next against this. It is for this reason that, after a half hour's stay in the Turkish bath, in a room heated up to 66° Cent. (or 151° Fahr.), the deeper-seated parts of the body never rise one whole degree of Centigrade scale, as I have myself ascertained by observations made upon a man, thirty years of age, into whose rectum I introduced the thermometer bulb while he was in the bath-chamber (I myself also being present). At the expiration of half an hour, I was forced to interrupt the experiment, for the man complained so much of the burning produced by the heated glass case which surrounds the stem of the thermometer. By staying in the Turkish bath, a sound individual may lose two kilogrammes (about five and one-third pounds) of water, as was shown by one of my assistants, who tried the experiment on himself, by weighing his own completely naked body both before and after the bath.

The requisite conveniences for carrying out this plan of treatment by hot-air baths ought to be forthcoming in all large hospitals where cases of dropsy are treated. In the Kiel Academical Hospital provision has recently been made for the erection of a bath-room of this kind. In a private house an imperfect substitute for the hot-air chamber may be contrived by seating the patient upon a wooden stool, in a well-warmed room, and putting one or two spirit lamps on the floor under the stool; after the lamps have been lighted, blankets should be wrapped round the patient in such a manner as to cut off the atmosphere immediately surrounding him from that of the room at large, his head alone being left exposed.

The so-called hot sand baths doubtless exert a similar effect to that obtained by the use of the dry hot-air bath; but I have never made use of them myself.

Persons suffering from heart disease should never be allowed to use the so-called Russian steam baths. It is true enough that a stay in the chamber filled with an atmosphere of aqueous vapor, heated up to 50° R. (145° Fahr.), will provoke profuse sweating; but then, since the surrounding atmosphere is saturated with moisture, no evaporation of water can take place from the surface of the body; the heat, therefore, developed by the tissue changes, which the high temperature has thus rendered excessive, must accumulate in the body, for this is not only unable to throw off any of its heat into the surrounding medium, but must absorb still more caloric from its wet hot enveloping atmosphere. In one man in our hospital, who used the vapor bath heated to 53° Cent. (122.5° Fahr.), I observed an elevation of temperature amounting to 3.6° Cent. take place in the rectum, while at the same time the pulse was raised to 170 and the respirations to 44—effects to which I should never venture to expose a patient suffering with heart disease.

To institute an effective diaphoresis, we are restricted, in the great majority of cases, to the prescription of prolonged hot baths at a temperature of 40° Cent. (104° Fahr.), or even upwards of this.

According to Liebermeister's[1] plan, the patient should be placed daily in a hot bath (at a temperature of 38° Cent. or 100.5° Fahr.), and the temperature of this should then be gradually raised up to 42° Cent. (107.8° Fahr.) by the addition of hot water; after the bath has been raised to this temperature, the patient should remain in it as long a time as he comfortably can (not beyond a full hour); the room being thoroughly warm, he should then be as quickly as possible packed in previously warmed blankets, and be kept wrapped up in these for one or two hours; he is then, finally, to be rubbed down quickly and put into a previously warmed bed.

Even cardiac cases bear this mode of treatment exceedingly well; still, until they are accustomed to it, it is as well to limit the duration of the bath to half an hour, and to direct the attendants never to lose sight of the patient while in the bath, that they may be ready to afford him the requisite help in the event of his being seized with faintness.

I have not yet instituted any observations upon the behavior of the corporeal temperature during the progress of this form of hot bath; it undoubtedly rises, but certainly not to any dangerous extent, as is the case under the Russian steam bath, where heat is supplied to the body, not merely through the skin, but through the lungs, by the inhalation of the hot vapor.

Less efficient than the above-mentioned means for producing diaphoresis, is simple packing in wet cloths; but this method is not to be despised when the opportunities of applying better measures fail us.

---

[1] Prager Vierteljahrschrift. Bd. 72. S. 1, et seq.

Where the diaphoretic method, however, fails to restrain the dropsy, there is no hope that better results will follow upon any other plan of treatment. I would expressly caution against too speedily resorting to mechanical measures for the evacuation of dropsical effusions. While, however, I consider paracentesis abdominis in excessive collections of fluid in the peritoneal sac as a safe and proper proceeding in cardiac, as well as in some other forms of dropsy, I cannot say the same of repeated scarifications of the swollen limbs. Notwithstanding the exercise of great care and cleanliness, we cannot always succeed in preventing the punctures from becoming the starting-points of an erysipelatous or gangrenous inflammation. Before proceeding to scarification, however, we ought to follow Bock's recommendation, and introduce a fine canula (like the hollow needle of Pravaz's syringe) into the dropsical limbs, so as to allow the fluid to escape gradually, drop by drop, and thus relieve our patient.

2. Ischæmia of the Kidneys and its Results. The Choleraic Affection of the Kidneys.

Ischæmia of the kidneys—that is, the more or less complete interruption or stoppage of the arterial blood-supply to the kidneys, occurring independently of simultaneous general hyperrepletion of the venous system—comes before us in practice only in the asphyxia stage of cholera, and in other conditions which interfere in a similar manner with the general circulation. For practical purposes, renal ischæmia may be considered merely as a symptom of cholera.

Attacks of cholera of the severer kind—let the interpretation of its cause be what it may, for it does not concern us—lead, without exception, to grave disturbance of the renal secretion, up to its complete suppression. That the drying up not only of the urinary secretion, but also of all the other secretions, in cholera is the consequence of the lowering of the arterial blood pressure, no one will dispute any longer, since Griesinger's investigation of the matter. Griesinger called attention to the fact that the anatomical changes, which are developed in the later stages of the disease, do not exist at all at the time when the complete suppression of secretion really commences, and cannot, therefore, be the cause of the disorder of function which exists. The interruption of secretion that takes place corresponds exactly with the results that follow upon artificial compression (up to occlusion) of the renal artery in animals.[1] The structural changes which the kidneys undergo in the cholera process are also identical with those that Cohnheim[2] observed in the kidneys of animals whose renal artery and vein he had firmly ligated upon leather and kept closed a certain time.

In point of fact, the cholera process at its height leads to complete stoppage of the circulation, like that which Cohnheim produced in a kidney artificially, in the experiment referred to above. Cohnheim, in his masterly treatise, gives us accurate information regarding the destructive results (in respect to the nutrition and structure) entailed by complete and prolonged interruption of the circulation, not in the kidney only, but also in other parts of the bodies of animals,—those parts, for instance, which are so organized that the changes which take place in them can be accurately studied for a considerable length of time. He shows that the grade of nutritional disturbance in the tissues thus involved corresponds directly with the length of time during which the circulation is interrupted. If this be quite short, the circulation is resumed quickly and without difficulty —indeed all the more easily, because the vessels into which the blood again has free entry at first become widely dilated. Transparent ears of rabbits, for example, are then seen to become fiery red in color. Gradually the vessels contract afresh, and, without evincing a trace of further change, the normal state of the parts is restored. But if the interruption continue for a longer time, when the ligature is removed, not merely do the vessels remain dilated, but there is swelling of the entire organ, or of those parts from which the blood supply has thus been cut off,—swelling produced by effusion of blood plasma through the walls of the vessels, and by the emigration, in large numbers, of red and white blood corpuscles through the walls of both veins and capillaries. If the auricle of a rabbit be ligated upon a cork stopper for some time, there ensues not merely a boggy œdema of the organ—the first result of a brief stoppage of the circulation—but even hemorrhagic infarction in its most typical form. Cohnheim saw a kidney swell up to twice the size of that on the other side, and become black-red colored both on its surface and in its substance; and under the microscope he found that the capillaries and other vessels of both the cortical and the medullary substances were very full of blood, while a moderate number of blood-cells occupied the interstitial tissues and the lumina of the urine tubes, principally the straight ones. The urine, too, was bloody.

---

[1] Compare *Max Herrmann*, Ueber den Einfluss des Blutdruckes auf die Secretion des Harns. Zeitschrift für rationelle Medicin. Dritte Reihe. Bd. 17, S. 1, ff.

[2] Untersuchungen über die embolischen Processe. Berlin, 1872, S. 47.

In the kidney, as in the case of the frog's tongue and the transparent ear of the rabbit, if the circulation is interrupted for too long a time, it is never resumed again; if the artery remain ligated beyond a certain time, necrosis sets in; and then, when the ligature is loosened, the organ remains flaccid and of a dirty gray color, and gradually softens down into a yellowish gray pulp.

Cohnheim states that the disturbances of nutrition which the protracted stoppage of the circulation through the tissues in question produces, are due to the injury which the walls of the vessels suffer in their substance; yet he was never able to discover the slightest alteration in the minute histological structure of vessels that had been thus a long while deprived of the contact of the blood that should have circulated through them.

If now we compare the effects of the cholera process upon the kidneys with those of Cohnheim's experiments upon the same organs, the identity of the two becomes self-evident. A short attack of cholera acts like arterial ligation of short duration; the secretion stops directly the blood pressure falls below a certain minimum; when reaction sets in, the arteries fill afresh and freely, the secretion returns, and all is normal again. But in the more serious cases, where the stage of asphyxia is somewhat prolonged, the secretion tarries its return; the first portions of urine passed are albuminous and often blood-stained; the vessels into which the blood passes once more are dilated, and, whereas, in their normal condition, they had resisted transudation, they now allow some portion of their contents to pass through their walls. In the gravest forms of the complaint, attended by protracted asphyxia, the urinary secretion is not resumed—not even after the pulse has become full, and the skin hot, and the re-establishment of the natural circulation elsewhere is plainly apparent.

That the comparison I have instituted between the effect of the cholera process upon the nutrition of the renal tissues and the results of temporary artificial interruption of the circulation is correct, is also proved by the disorders of nutrition which are occasionally found in other organs in the bodies of those who die of cholera, affording appearances which correspond entirely with those obtained by Cohnheim in his experiments. For instance, I may call attention to the hemorrhagic infarctions which, in protracted cases of this disease, are often found in the lungs and spleen, as well as the patches of enteritis which advance nearly to gangrenous destruction. That the kidneys suffer more commonly than other organs from inflammatory changes after an attack of cholera, may well depend upon the conditions under which the circulation is carried on through these organs, and the peculiar arrangement of the vessels in the cortex, which proffer great opposition to the forward current of the blood,—a state of affairs, therefore, which, in the presence of general depression of the arterial pressure, will lead to complete interruption of the blood current more easily in this than in any other network of capillaries in the body.

Consideration of this peculiar distribution of the renal vessels—the formation of the marvellous network that lies between the vas afferens and vas efferens of the glomerulus and the breaking up again of the latter into a secondary capillary network—compels us to recognize notable differences between the effects of complete closure of the renal vessels by ligature and the operation of the cholera process. Thus ligature will cut off the circulation quite equally from all the vessels of the kidney, whereas the cholera process will affect first and foremost the vessels of the cortex. This difference, perceptible in the changes first operated, corresponds with what may be seen later on in the ultimate results attained by the two modes of procedure. While Cohnheim, in the course of his experiments, noticed that an equal dilatation took place in the vessels of both the cortex and the medulla, cholera leads principally to nutritional changes in the cortex. The opposite opinion, held by Virchow, that cholera nephritis should be regarded as the extension of a catarrh from the pelvis of the organ upwards through the papillæ, although accepted both by Reinhardt and by Leubuscher, has not been confirmed by later researches.

But between the two processes referred to above, a wider difference obtains—one doubtless founded upon the circumstance, that when the arterial ligature is loosed, the blood streams back again suddenly, with the full force of normal arterial pressure, into the vessels from which it has been excluded; whereas the circulation, after cholera, is at first only gradually re-established. How far the dissimilarity thus obtaining in the two processes explains the features of unlikeness in the results, we are not yet in a position to decide.

The Morbid Anatomy of the Kidney in Cholera.

In persons who have died of cholera, the kidneys exhibit very different appearances, according to the stage of the disease at which death has been induced.

If the patient succumb at the stage of asphyxia, the kidneys, as Reinhardt and Leubuscher[1] found, are not particularly enlarged; the vessels—the veins chiefly—were sometimes slightly hyperæmic, but that marked capillary injection, observed in a later stage, was never noticed by them in this early one. But already in those who died asphyxiated, they often found faded patches in the kidneys, spots that looked white or yellow; their interpretation of these thus altered portions was, that the renal tissues had been infiltrated with exudation, and they thought that this deprivation of color manifested itself first in the pyramids. It was for this reason that they adopted the theory put forth by Virchow, and mentioned briefly above.

---

[1] Virchow's Archiv. Bd. 2, S. 496.

Buhl[1] found the kidneys, after an attack of cholera of only twelve hours' duration, shrunken, firm, tough, and presenting, upon section, a dark, brown-red, somewhat livid, color; and dark blood dripped from the cut surfaces. I, too, in a similar case, have seen the kidneys looking smaller than normal.

Reinhardt and Leubuscher found, upon microscopical examination of these faded patches in cholera kidneys of the first stage, that the epithelial cells of the urine tubes were so firmly adherent to one another that upon pressure they could be separated from each other only by the use of much greater force than is ordinarily required. The epithelium, too, in these spots was cloudy and more opaque than elsewhere. Already at this same stage the lumina of the urine tubes were not uncommonly found blocked up with exudation; and when the algid condition had endured for a considerable time, firm cylindrical deposits were found in the tubuli uriniferi, and especially towards the apices of the pyramids.

Ludwig Meyer[2] has followed up these investigations of Reinhardt and Leubuscher upon the renal anatomical changes induced by cholera. He brings into prominent notice the peculiar stickiness of the kidney, found even after a very rapidly fatal attack of cholera; "it is as if the kidneys were saturated with albumen." He, too, like Reinhardt, describes, as characteristic of the cholera kidney, the extraordinarily early fatty metamorphosis of the epithelium and the exquisitely diffuse form of the disease, so that in one and the same fine section, prepared for microscopical examination, and taken from the cortical substance, tubes that were more or less highly fattily degenerated, and sprinkled with hemorrhagic foci, lay close to bundles of tubes whose contents appeared to be normal. Meyer, however, does not mention noticing the departure of these changes from the pyramids as starting-points, and Kelsch[3] denies Virchow's doctrine positively.

---

[1] Zeitschrift für rationelle Medicin. 1855, S. 47.

[2] Beitrag zur Pathologie des Choleratyphoids. Virchow's Archiv. Bd. 6, S. 471.

[3] Revue Critique et Recherches Anatomo-physiologiques sur la Maladie de Bright. Archives de Physiologie Normale et Pathologique. 2me Série. Tome I. 1874, p. 722.

As to the renal changes developed later on in the course of the disease, after the stage of reaction has set in, all observers are agreed that these involve the cortical substance by preference; this part swells up, and the whole kidney thus becomes greatly enlarged beyond its normal volume. The organ obtains a gray-white color, and is more easily torn than it should be. But even at this stage the unsymmetrical manner in which the changes spread is quite noticeable, the pyramids retaining for the most part their normal coloring close by gray-white portions of cortical substance; certain spots, however, of the cortex assume a bright yellow coloration, from accumulation of a quantity of fat in the epithelium. Where the process is further advanced, the urine tubes are much distended, and appear, under the microscope, completely opaque to transmitted light, while here and there the enlarged epithelial cells are broken down into a fatty detritus.

Reinhardt, whose description I have followed in this account of the cholera kidney, finds it remarkable that comparatively so very little hyperæmia of the organ should precede these changes, and that fatty degeneration should take place in the cells thus attacked within so few days from the commencement of the disease. Other observers found fatty degeneration of the epithelium within twenty-four hours from the date at which the process of disease began. Buhl[1] says, when the change has advanced to its fullest, the cortical substance is seen to be reduced to a white pulp, creamy or pus-like, although its microscopic features show almost nothing but fat.

Ludwig Meyer[2] was the first to show how perfectly entire the Malpighian capsules and the capillary coils of the glomeruli remain, even when the renal epithelium has undergone serious changes; and this circumstance has similarly been attested to by every later observer.

From the accounts given by Reinhardt and Leubuscher of what they found, I may call especial attention to the presence of hemorrhagic infarctions in the cholera kidney. They had already seen some small infarctions in the kidneys of a person who died in the algid stage; with this exception, however, these changes were found either in those who had died in the typhoid stage, or else of complications in the stage of reaction. The infarctions were without exception seated in the cortical substance, and in one instance were so extensive as to occupy more than two-thirds of the cortical substance. Ludwig Meyer, too, mentions a case of extensive hemorrhagic infarctions of the kidney aftercholera.

---

[1] l. c. S. 58.

[2] l. c. S. 490.

The accounts of writers are silent upon the state of the intertubular tissues; Kelsch only states that no change can be detected in them.

The mucous membrane of the urinary passages, the renal pelves, the ureters, and the bladder is found swollen and coated with a yellow, pus-like material, formed of desquamated epithelium, mucus- or pus-cells, and in the later stages of the disease it is hyperæmic, especially about the neck of the bladder, where ecchymoses may even be formed.

Now, how shall we explain the changes, above described, which the kidneys of cholera patients present to us? Reinhardt and Leubuscher regarded them as an inflammatory infiltration. Virchow had previously described them as catarrhal nephritis. Frerichs describes them as a peculiar form of Bright's disease. Ludwig Meyer, nevertheless, is unwilling to accept Reinhardt's interpretation of the renal changes of cholera, viz., that they are the consequence of inflammatory irritation due to some peculiarity in the composition of the blood induced at this stage of the complaint. He says: "In the first place, the appearances presented by the special secreting structures themselves, which would be first and most exposed to the action of any such irritant, speak at once against the adoption of this idea, which indeed is still purely hypothetical in its nature; and then, in the next place, all the conditions fit in so entirely with the supposition of a disturbance of the circulation—such as actually exists—as the cause of the renal changes, that we may almost look upon this view as established to a certainty. One needs merely to glance at the symptoms of asphyxia, and at the bodies of those who have died thus asphyxiated, to possess an all-convincing picture of this blood stasis in the capillaries and entire system of veins. The stagnating blood allows a part of its serum to diffuse itself through all the tissues, soaking them with a solution of albumen; the skin becomes boggy, the muscles sticky and stiff; the serous membranes are covered with a layer of albumen, which bestows on them a soapy feel, and makes them glide very easily out of one's hand; the kidneys present the same characters."

Who, then, perceiving the direct correspondence between this account of the facts observed and the results of Cohnheim's clever experiments in the temporary interruption of the blood-current through organs that admit of being watched the while, but would be convinced by them? The only difference lies here, viz., that Cohnheim has placed a different interpretation upon the process which he was able to follow step by step throughout its whole course, from that which Meyer has presented us in explanation of the changes found in the cholera kidney. It is not the stasis of the circulation through the veins (a thing, moreover, which exists originally no more in the cholera kidney than it does after ligature of the renal vessels) that induces the dilatation of their walls and the alteration of their permeability; these changed conditions are directly due to some disturbance in the physiological functions of the vessels. The first event that happened when Cohnheim loosed the ligature of the vessels was the dilatation of them all; shortly afterwards the arteries began to contract again. But not so the veins; these remained dilated; and, watching them attentively, one had not long to wait before evident and abundant migration of the colorless corpuscles began from all the small and medium-sized veins; and, self-evidently, there was also an escape of blood plasma, which made the rabbit's ear thus treated swell up to the thickness of a centimetre. Migration of the colorless corpuscles also tookplace from the capillaries; but here, as Cohnheim observed, red corpuscles made their way through at the same time (per diapedesin).[1]

---

[1] l. c. S. 36.

Kelsch also did not fail to perceive the correspondence between the condition of the kidneys following cholera and that which was produced by artificial interruption of the renal circulation. He is unwilling to admit, however, that the above-described renal changes partake of the nature of inflammation, because he is unable to find in these organs under such circumstances the essential proof of an inflammatory process, viz., cellular infiltration of the interstitial tissues. At the same time he denies the existence of any kind of hyperæmia; and his inability, therefore, to find evidences of an inflammatory process loses much of its value, since, while the German writers fail to make any mention of the state of the intertubular structures, all are agreed that venous hyperæmia follows regularly upon the renal changes induced by cholera. One might suppose that Kelsch's investigations of the cholera kidney had been pursued at that stage of affairs, or at that time, when the migrated colorless cells had been already taken up by the lymphatics, a proceeding which Cohnheim was able to follow directly in his experiments upon the frog's tongue, and which took place under favorable circumstances very quickly. Kelsch attributes the swelling of the white cholera kidney to a necrobiotic destruction (disintegration) of the epithelial cells, brought about by the ischæmia.

It is impossible for me to understand how the kidneys, by a simple cutting-off of their nutritious supply—as Kelsch maintains—can become larger and heavier, or how, without the intervention of an increased blood supply, a new formation of epithelial cells can take place in the cholera kidney in the room of those which have been removed by necrosis—a regeneration which Kelsch himself has observed.

For myself, I have no hesitation in saying of the process which takes place in the kidney after cholera the same that Cohnheim said in regard to the pathological changes which the rabbit's ear suffers in temporary interruption of the circulation through it: "Now, if these changes be not inflammatory in their nature, and the whole process do not admit of being regarded and described as inflammation of the ear, I should certainly like to know what we are to understand by the term inflammation."[1] I should therefore, in a certain sense, have been more correct in describing the choleraic renal affection in the following section of my work, which treats of acute parenchymatous nephritis, if the state of kidney leading up to it had not been accompanied by disorders of function, which were in themselves quite peculiar. Moreover, the further progress of the inflammation of the kidney which follows cholera differs so essentially from all forms of renal inflammation of other origin as to establish its claim to a separate notice.

The further course of this affection, for instance, is entirely analogous to those processes seen and described by Cohnheim in the parts of animals upon which he experimented. Just as, after too protracted ligation of the renal vessels, he observed that the whole kidney became a gray-yellow fawn-colored pulp, so Buhl found that the cholera process, after it had advanced to its furthest extent, left the cortical substance of the kidney in some places pale and pultaceous—although, of course, changes so extensive as this would always be rare, since death, for the most part, anticipates their development.

In cases of recovery, quite like what happens after a temporary artificial interruption of the circulation, the functional disturbances effected by the cholera disappear after a little while, and the anatomical changes subside as well. The albuminuria and the excretion of casts—the evidences of the anatomical changes impressed—last but for a few days, and then complete restoration to a healthy state follows, just as, after speedy removal of the ligature from the rabbit's ear, the morbid changes entailed soon entirely disappear. I know of no instance in which the passage of this choleraic affection of the kidney into a chronic renal disease, in any way akin to morbus Brightii, has been affirmed with certainty; the two cases of Hamernik's, cited by Frerichs, cannot be accepted as examples, since from Hamernik's own[2] scanty account of them it remains by no means certain that the individuals affected had not had renal disease before

---

[1] *Dr. Julius Cohnheim*, Neue Untersuchungen über die Entzündung. Berlin, 1873, S. 67.

[2] Die Cholera epidemica. Prag, 1850, S. 125 u. 126.

they had cholera, as happened in one of the patients in my own clinical ward. Indeed, the first of Hamernik's cases scarcely admits of any other interpretation.

The woman's attack of cholera was so slight that she was able to go about her duties in the house. When the diarrhœa ceased, the patient had an extreme urticaria, for which she came into the hospital. "The urinary secretion the while was abundant enough, the urine was pale, and contained a moderate amount of albumen."

After a lapse of rather more than fourteen days, the patient had become dropsical; by degrees the dropsy steadily increased; uræmic symptoms and death ensued. "At the autopsy the ordinary appearances of Bright's disease were discovered."

Of the second case it was likewise stated that the cholera affection was so slight that the patient "was able to get about the greater part of the time." When, however, the evacuations ceased, albumen remained in the urine; the patient became gradually dropsical, and in a few weeks from her illness died, with the ordinary symptoms of Bright's disease.

No post-mortem appearances are communicated.

From these scanty details it is by no means clearly established that the patients were recovering from real cholera.

According to Meyer's idea, the kidney affected by cholera returns to its normal state by a fresh formation of epithelium in the place of that which has been destroyed. He describes cells which attracted his attention in his examination of the kidneys belonging to later typhoid stages of cholera. These were smaller than the normal renal epithelium, rather oval in shape, and provided with long, oval nuclei. Later on he was fortunate enough to find whole portions of urine tubes lined with cells of this kind, some of which appeared in process of transition into forms of the ordinary shape.[1] Buhl, too, speaks of regenerated epithelium. This new formation of epithelial cells, to take the place of those which were cast off after the attack of cholera, has quite recently been confirmed by Kelsch.

Symptoms.

The functional disorders manifested by the kidneys during the cholera process stand in the closest relation to the severity of the attack and the length of its duration. The milder forms of cholera affect the function of the kidneys no further than any profuse excretion of fluids from the intestines might influence it; the secretion becomes scantier, and the urine voided correspondingly more concentrated. It is highly probable that, even in cases of well-pronounced vomiting and diarrhœa, so long as the pulse can be distinctly counted, and the skin remains in some measure warm, the urinary secretion will not be entirely suppressed, although the bladder may not empty itself once in the twenty-four hours. In such cases the urine frequently does not exhibit a trace of albumen, and shows no casts; at all events, I have never found casts in the non-albuminous urine of cholera patients in whom the urinary secretion had not been long arrested, and who, as a matter of course, had never previously passed albuminous urine. On the other hand, I have had a number of cases where no urine at all was secreted for several days, although the pulse could be distinctly felt at the wrist.

---

[1] l. c. S. 497.

Should a genuine condition of asphyxia with pulselessness supervene, the secretion of urine ceases, as a rule, entirely, and is, in most cases, not established again, since patients in this condition mostly die. But should the algid stage be succeeded by that of reaction, the arteries then fill afresh, the warmth returns to the skin, and the secretion of urine too generally is restored; the return, however, of the secretion of urine will be delayed in proportion to the length of time the stage of asphyxia has endured. Several days of absolute anuria may thus pass away. Now, if the urinary secretion is entirely interrupted for only a short time, upon its first re-establishment a very small quantity only is secreted, and this small quantity, in far the larger number of cases, contains albumen; albumen, however, is invariably present when the secretion of the kidneys has been for some days completely suppressed.

As to the other characters of the urine, first secreted anew after an attack of cholera—its specific gravity, quantity, composition, etc.—the accounts given do not quite harmonize one with another, and this apparently for the reason that the portions of urine first passed follow sometimes earlier and sometimes later after the date of resumption of the renal functions, and also because the urine that has been examined has been collected at different periods after the re-establishment of the secretion, often at widely different periods, and must therefore have furnished varying results in the several instances. As a general statement, we may say that after an attack of cholera the quantity of urine passed is, for a while (one or two days), scanty. In one of my cases, however, which ran on to a fatal issue in the course of three days, and in which, in spite of well-marked cyanosis, the pulse remained remarkably strong, the secretion of urine (containing at first an abundant, and later on a small quantity of albumen) was so far maintained that a daily average of 1,000 c. ctm. was passed. Ordinarily the secretion, when once interrupted, returns at first by degrees. The most accurate observations upon the urine of cholera emanate from Buhl[1] and Dr. Oscar Wyss.[2] Buhl's investigations upon the time of restoration of the urinary secretion after cholera rest upon the data of 133 cases. In fourteen cases, urine was passed between the eighteenth and twenty-fourth hours, in forty-one upon the second day, in forty-seven on the third day, in thirty on the fourth day, in seventeen on the fifth day, and in five upon the sixth day. If by the sixth day no urine had been passed, it never was, for death ensued. The results of Wyss's investigations confirm essentially those of previous observers: in the first twenty-four hours after an attack of cholera, from 100 to 200 c. ctm. of urine are passed; then, if reaction set in vigorously, the quantity of urine rapidly increases, so that upon the second day, in favorable cases, the total may rise up to 400, or even, in some cases, to 800 or 900 c. ctm. The following days the quantity still continues to increase, and in the milder cases reaches its maximum between the fifth and sixth days, although in the graver cases this may be postponed till later, and in one case, for example, narrated by Wyss, was only reached on the tenth day. This maximal quantity can be as much as 4,000 or 5,000 c. ctm., as Goldbaum had already stated it to be; but the excessive urinary secretion then gradually sinks down again to the normal. The sequelæ and complications that may set in in the stage of reaction are able to disturb this hypersecretion of the kidneys or even to arrest it; anuria may occur once more, and this is then usually fatal.

---

[1] l. c. S. 24.

[2] Ueber die Beschaffenheit des Harns im Reactionsstadium der Cholera Asiatica. Archiv für Heilkunde. 1868, S. 232.

The specific gravity of the urine first secreted after an attack of cholera is found in different cases to be exceedingly diverse. Wyss, who carefully tested the matter, found it to vary between 1012 and 1033. But all observers are entirely agreed that the specific gravity of the urine diminishes as its quantity increases, so that it may sink as low as 1005, and upon the fifth day after the restoration of the secretion, and later on, may fall even lower, rising, of course, again to normal as convalescence progresses.

The reaction of the urine is at first acid. The color of that passed first, after an attack of cholera, is red-brown or red, rarely pale yellow. Even the earlier observers had noted the fact that the urine of cholera altered its color in a remarkable way when treated with the mineral acids; some, in consequence, had arrived at the idea that the coloring matter of the bile was pretty constantly present in cholera urine. Wyss's researches convinced him that the peculiarity which cholera urine had of becoming dark-colored by decomposition, or upon treatment with mineral acids, rested on its remarkably large content of indican, which, under the above-mentioned influences, was converted into indigo. Wyss further found true bile pigment in one case in the first urine secreted; in this instance, however, there was no icteric tint of the skin.

The first post cholera urines, as I have seen myself, are invariably cloudy. Casts are always present in the sediment, and these, as Meyer has pointed out, are of a quite remarkable length, both thick and slender, and are usually present in large quantity. Wyss considers it to be a favorable sign when even the very first urine, passed after the seizure, contains a large number of casts; he found that those cases in which the urine remained poor in casts were always severe and usually fatal ones. Sometimes the excretion of casts lasted for only two days. Wyss lost sight of them repeatedly on and after the sixth day; whereas, in other cases, he was still able to find them on the eleventh, twelfth, and thirteenth days.

Various kinds of casts are found in cholera urine. According to my own observations, far the most predominant are the perfectly pale and homogeneous cylinders, the so-called hyaline forms; but when the excretion of casts has lasted for some little time, these become dotted in parts with fine fatty molecules. I interpret them, therefore, to be fibrinous coagula, or at all events the products of precipitation from albuminous urine. Finely granular or fatty renal epithelial cells, or broken fragments of cells together with their nuclei, are very usually found sticking to these casts. Renal epithelium, however, is present separately, although there is not much of it. Besides these I have repeatedly seen some peculiar forms in cholera urines—forms which Thomas, in his previously quoted work upon the renal affection of scarlet fever, has described as cylindroids. Ludwig Meyer[1] and Wyss[2] have obviously had the same objects before their eyes in their examinations of post cholera urine. Wyss speaks of having seen quite dark, granular, crumbling casts, associated with highly refractive, sometimes pale, straw-colored forms. In rare cases I have seen specimens of the former variety, but do not remember ever to have seen the latter in cholera urine. Wyss mentions the fact that he once found casts in the first urine passed, without any albumen being discoverable.

Blood-corpuscles are certainly not always present in the first specimens of urine secreted, although occasionally they occur in considerable quantity. Wyss even saw small blood-coagula. But we must recollect the ecchymoses which occur in the mucous membrane of the bladder, and only attribute the admixture of blood-corpuscles to the kidneys when blood-casts are also present.

Of the other formed elements discovered in cholera urine, it is only necessary to mention the epithelium from the urinary passages—which Wyss found to contain an unusual amount of fat-globules—and pus-cells in greater or less abundance; the latter probably consist, in part, of white blood-corpuscles which have migrated from the vessels inside the kidneys, and in part of the products of catarrh of the urinary passages. The presence of urates, of octahedral crystals of oxalate of lime, etc., in the sediment, is of no particular importance.

---

[1] l. c. S. 486.

[2] l. c. S. 244.

Now, except in the milder cases, in which complete suppression of urine never takes place, and in which the renal secretion either never contains albumen at all or exhibits only temporary traces of it, the fact may be assumed as an invariable one, that the first urine passed after a well-marked attack of cholera does contain albumen, and this, too, for the most part in considerable quantity.

Unfortunately, I failed to avail myself of the only opportunity I ever had of estimating the quantity of albumen contained in cholera urine; the occasion was one in which I and my assistants were overwhelmed with other work to accomplish, and these inquiries, which would have engaged so much time, were forced to be left undone.

The most highly albuminous urine which we have ever encountered contained, as was estimated, nearly two per thousand,—certainly an insignificant quantity, if one compares it with that contained in the urine in many cases of nephritis. Wyss, too, never made any exact estimates of the albumen in his investigations. According to my own scanty experience, which tallies entirely with that of Wyss, the urine first passed contains the largest amount of albumen. In the milder examples of the complaint, one occasionally fails to find albumen after the second day; the albuminuria lasts, as a rule, from five to eight days, and almost never continues beyond the end of the second week.

The percentage content of urea in cholera urine naturally varies greatly, fluctuating with the specific gravity. It stands to reason that the absolute quantity must be very small, considering the small amount of urine secreted,—in the first days reaching to only a few grammes,—and it is only the abundant secretion of the succeeding days that leads to its excretion in large quantities. In this way, according to Buhl, the faulty elimination of the nitrogenous products of tissue waste, brought about by the interruption of the renal secretion, is finally completely made up. This compensation, however, follows all the slower, the longer the complete suppression is maintained.

Like the urea, the other normal constituents of the urine are present only in small quantity in the first urine passed after an attack of cholera. But in the succeeding days the quantity of these other salts is not increased in the same proportion as is the urea, which has been stored up in the tissues and the blood throughout the attack. The explanation of this is that these other (blood) salts, and especially the chlorides, are carried off pretty freely from the blood during the cholera by the intestinal discharges.

It is also worth noticing that occasionally, in the course of convalescence, sugar appears intermittently in the urine, and in quantities which are somewhat considerable. Wyss, who carefully investigated this post cholera glycosuria, discovered sugar in the urine between the fifth and eighth days in a relatively large amount.

Upon the etiology of the choleraic disorder of the renal functions I have expressed myself enough above. Meyer, Buhl, and Griesinger attribute the interruption of the secretion solely to the disturbance of the circulation. As further proof of the correctness of this view, I might direct attention to the results of Herrmann's [1] experiments upon the effect of narrowing the renal arteries in living animals by means of a compressor; the secretion of urine was thereby arrested directly the blood pressure fell to too low an ebb.

No one really has disputed the view that interruption of the circulation is the cause of arrest of the secretion of urine in cholera; but it is far otherwise in respect to the opinions entertained as to what causes the delay in the return of this secretion after the re-establishment of the circulation. The earlier observers clearly were inclined to attribute this retardation, or even complete suppression, of the secretion of urine, that occurred in the stage of reaction after cholera, to inflammation of the kidneys, or to blocking up of the urine tubes with the products of inflammation. They failed, however, to appreciate the fact that the inflammatory renal changes do not take place during the attack of cholera, but appear first in the stage of reaction. Buhl[1] had already said: "The cause of this suppression cannot be any anatomical obstruction of the passages, for the first and second outflows of urine, even when these do not occur for the first time until the sixth day, must completely clear the passages of any obstruction in them, and the second or third discharge would meet with scarcely any further fibrinous or epithelial plugs to obstruct its way; further, the albumen ceases and the Malpighian vascular tufts too remain perfectly intact. The cause, therefore, lies solely in the interruption of the capillary interchanges provoked by the attack of cholera in the kidneys, as well as in other organs." This interruption of the capillary interchanges in every tissue of the body he describes as a kind of trance or apparent death. How completely Buhl's views of the matter, so far as the vessels are concerned, are justified by Cohnheim's experiments, I have detailed above.

---

[1] Ueber den Einfluss des Blutdrucks auf die Secretion des Harns. Zeitschrift für ration. Medicin. Dritte Reihe. Bd. 17, S. 1 ff.

If now we turn our attention to the effect upon the entire body of this interruption of the renal secretion in cholera which we have described, the following facts impress themselves on us. The circulation, if interrupted too long, never re-establishes itself, and then there is no further return of the urinary secretion; or the circulation is re-established after some halting, and is associated with extreme disorders of nutrition in the kidneys, dependent on the abnormal permeability of the walls of the veins and capillaries for their contents. Under these circumstances the renal secretion remains deficient, not sufficing to carry off the nitrogenous products of tissue waste which have meantime collected in the system. Under both conditions death may be ushered in by uræmia. The nutritional interchanges, it must be remembered, do not stand still during the cholera process; indeed, upon the advent of the stage of reaction, they are increased above the normal, as is proved by the elevation of the bodily temperature. Were now the excretion of the nitrogenous products of tissue waste to be completely arrested, these must of necessity remain stored in the fluids and tissues. So far back as 1850 Hamernik[1] avowed that in the graver cases of cholera, where no urine was passed, he had perceived that the sweat, which was abundant and sticky, and had the odor of urine, if collected from the forehead and nose and cheek-bones, deposited a sediment which evaporated down to a greasy powder, in which uric acid salts and fat could be found. Schottin[2] observed that enormous quantities of crystalline forms were excreted upon the skin of the face, as well as on that of the upper part of the chest and arms, in cholera patients just in the agony of death, and these he made out to be masses of urea. It is possible that Hamernik's observation rested upon similar data, and that his interpretation of them only was erroneous.[3] Later on Buhl discovered that the blood of cholera patients contained enormous quantities of urea. The blood of a girl who died in the typhoid stage of cholera (on the ninth day), with symptoms of opisthotonos, gave him 0.2 per cent. of urea.

---

[1] l. c. S. 82.

We see, therefore, that while cholera may not at the outset lead to a fatal collapse, the suppression of the renal functions which the attack brings with it may lead to death, and this by uræmia. Death, too, under these circumstances, follows before any dropsy can occur from the arrest of the urinary secretion. Cholera probably never terminates in dropsy. The cause of the suppression of the secretion of urine is certainly the enormous drain of water to which the system is subjected by the vomiting and the diarrhœa, and by which the vessels are robbed of the greater part of their contents and all the tissues are dried up. But if now the choleraic discharges cease, and water is taken up again into the blood from the intestines, so that the tissues once more obtain their normal grade of moisture, all the excretory materials that have collected in the tissues are swept into the circulatory system again, and in the favorable cases are forthwith excreted from the system by means of the excessive secretion of urine. Should, however, this customary excessive secretion of urine fail, the retention of the excretory substances may provoke a fatal attack of uræmia, to which some of the cholera patients, who have been so fortunate as to escape the first perils of the attack, succumb. It is, of course, perfectly correct that every case of so-called cholera typhoid is not the result of the retention of urinary elements in the system, as was once assumed; the implication of other organs in processes of disease as sequelæ of the cholera process—as, for example, the dysenteric-like, necrotic affection of the intestinal mucous membrane—can import very serious typhoid symptoms into a case where the renal functions have been tolerably well restored. Still, there will always remain some cases whose fatal issue, after reaction has set in, can only be attributed to either total suppression of urine or its incomplete re-establishment. In such cases every symptom of acute uræmia may be presented. But, speaking generally, epileptiform and spasmodic attacks are not very common after cholera, as the experience of all who have had much to do with cholera teaches us. It is far more usual for a condition of coma to supervene, without precursory cramps. In October, 1873, a laborer, thirty-eight years of age, the subject of granular atrophy of the kidneys, and who had had the good luck to survive a series of attacks of uræmic convulsions just a year before, succumbed to cholera. The last four days of his life he did not pass a drop of urine, and died in the profoundest coma, without the slightest previous sign of convulsions.

---

[1] l. c., S. 211.

[2] Archiv für physiologische Heilkunde. Bd. 10. S. 469 ff.

[3] That is, urea, and not as he supposed, the urates.—TRANS.

Diagnosis.

So far as the diagnosis is concerned, it is only necessary to establish the fact that the patient is suffering from cholera; and this is not the place for me to dilate upon this subject.

Prognosis.

As regards the prognosis in any particular case, the degree of activity of the renal functions is of very great importance, and it is only to the determination of this and the establishment of its value in prognosis that I need address myself.

According to all experience, the rapid return of the renal secretion after a severe attack of cholera is a very favorable symptom ; and, contrariwise, the peril of the patient's life increases with every day that the suppression of urine is protracted : first, because this retardation appoints an incomplete re-establishment of the circulation ; and, secondly, because the risk of the kidneys becoming incapable of resuming their functions increases with each day that these are abrogated. If by the sixth day no secretion has returned, Buhl's experience is that it never does, and that the patient's doom is sealed. But, even when the secretion returns at an earlier day, danger is still to be apprehended from the kidneys when the daily quantity of urine passed is an abnormally low one ; when by the fifth or sixth day a very abundant secretion has not been established, or, after being established, has sunk afresh far below what is normal. If the urine is once secreted in full flow, the patient's risk, so far as his kidneys are concerned, is overpast. Every trace of former disturbance in the organs themselves is most rapidly effaced ; no chronic renal affection ever remains as a sequel to the attack.

The percentage quantity of albumen contained in the first urine is of no particular prognostic import. I have seen cases, where the first urine was loaded with albumen, rapidly recover ; and others, in which the fluid first secreted afforded scarcely a cloud by heating, terminate fatally.

Whether the occurrence of any great number of casts in the first urine can be considered a favorable omen, as Wyss has assumed it to be, I am not able myself to decide.

Treatment.

The treatment of renal ischæmia and its consequences coincides with that adapted to the attack of cholera, and this up to the present day, for lack of a better one, and apart from any measures of prophylaxis, remains merely the treatment of symptoms as these arise. Since every endeavor to restrain the prodigious drain of water from the intestinal tract by direct means has hitherto proved fruitless, we should strive, on the one hand, to distend the capillaries of the skin by hot baths, and by this means to divert the blood stream that is rushing towards the intestines, and, on the other, to assist the failing action of the heart by stimulating it. Such stimulants as can be swallowed do not exert much effect, since they are either rejected by vomiting or fail to be absorbed at the right time. I think I am justified in attributing good effects to a method of stimulation recommended to me by the late Dr. Lindwurm, and which consists in the subcutaneous injection of a decigramme (one and a half grains) of camphor dissolved in a gramme (fifteen drops) of olive oil, a measure which may be repeated every three or four hours, or more or less frequently, according to the nature of the case.

In desperate cases I should certainly resort to the injection of warmed solutions of salt into the veins, as first essayed by English observers of cholera in India. This method has lately been resumed in London by Dr. Louis Stromeyer and Dr. Little, with satisfactory results.[1] Two cases in which I tried it certainly remained unbenefited ; but this may have been, perhaps, because I operated too late.

These, after all, are, so to speak, merely measures of prophylaxis, by which we endeavor to anticipate the dangers that attend the cholera seizure, and thus prevent the occurrence of the graver nutritional lesions of the kidneys.

Once the cholera attack is passed over and reaction has set in, if the urinary secretion is still delayed, we know of nothing that will establish its return. Stimulants, if the heart be failing, and warm baths to further the circulation of the blood, may well be resorted to ; but we know of no way by which we can work directly upon the paralyzed state of the renal vessels.

3. Parenchymatous Nephritis.

a. The Acute Parenchymatous Inflammation of the Kidneys.

The acute parenchymatous inflammation of the kidneys attracted the attention of physicians long before they became acquainted with the chronic, diffuse, or disseminated forms of renal disease; at least they observed that certain changes took place in the urinary secretion in this form of the disease, and referred them with greater or less certainty to certain anatomical conditions of the kidneys. Even in the last century accounts were written and observations made by medical men upon dropsy and hæmaturia after scarlet fever. Wells directed special attention to the character of the urine after scarlet fever, and showed that this secretion, even when uncolored by blood, might still contain albumen.

---

[1] Clinical Lectures and Reports of the London Hospital. Vol. IV. 1867–'68, p. 431 et seq.

Fischer[1] appears to have been the first person who admitted the existence of inflammation of the kidneys after scarlet fever.

Bright himself, in his first publication, does not mention the influence which the scarlatinal process may exercise in originating renal disease. But Hamilton, as early as 1833, reported that he had found the post scarlatina kidney, even when no dropsy had existed, in just the same condition as in the first stage of Bright's disease. Christison, too, mentions scarlet fever as a "predisposing, if not even the actual exciting cause" of granular degeneration of the kidneys (a designation under which he included every diffuse affection of these organs), and adduces two cases, to one of which a short account of the post-mortem conditions is appended.

P. Rayer, however, was the first to recognize and distinguish, clearly and definitely, an acute from a chronic form of what he called *albuminous nephritis;* he also recognized the fact that the acute does not by any means always pass into the chronic form, but usually only under those circumstances when the affection is left wholly to itself. He described the symptoms and the course of acute nephritis, and attributed it in children to scarlet fever, in adults to the effect of sudden changes of temperature, especially to cold and moisture.

Later German writers maintained that acute parenchymatous nephritis passed on into the first stage of their morbus Brightii.

---

[1] Journal für praktische Heilkunde, herausgegeben von *C. W. Hufeland* und *E. Ozann.* Februar, 1824.

Johnson calls it acute desquamative nephritis, and apparently allows it to originate from every cause which can give rise to renal disease. Traube names it[1] hemorrhagic nephritis; but his description of its morbid anatomy and its course does not seem to me to correspond with the reality.

In the most recent German handbooks and students' works of reference it has obtained sometimes the name of catarrhal, and at others that of croupous nephritis; and at the same time some of the cases are reckoned among the chronic forms of parenchymatous nephritis.

Lecorché treats of it as a "néphrite parenchymateuse superficielle ou légère."

Etiology.

The unmistakable causal connection between acute inflammation of the kidneys and certain processes which take place in the skin did not escape the notice of even the early observers, and as a result all sorts of hypotheses were suggested in explanation of the connection between the disturbance of the functions of the skin and the renal inflammation that succeeded it. The well-known figure of speech, however, which represents a certain antagonism between the skin and the kidneys, affords us but little assistance in interpreting the facts; and so far as the irritant substances are concerned, which, it has been assumed, remain behind in the system directly the secretions of the skin are arrested, and irritate and inflame the kidneys in the process of their elimination by these organs, not one has hitherto been found.

Acute parenchymatous inflammation of the kidney occurs, too, as admits of demonstration, under circumstances where there can be no question of its having been caused in any measure by a previous skin affection. Nevertheless, the fact is well established that a great number of these cases either succeed some impression that has been made upon the skin, or follow some pathological alteration of the general epidermoid covering.

---

[1] Gesammelte Beiträge. Bd. II. S. 1029.

To one who impartially observes the matter, it will certainly be apparent that the relation between the changes which take place in the skin and in the kidneys is not of a simple nature; both the cutaneous and the renal alterations are, in many cases, no other than mere co-results of some unknown third or main cause that has provoked them both. Acute nephritis can originate in various different ways, and I think it is worth while—in order to arrive, as far as possible, at a clear understanding of the causes which, according to our experience, most commonly provoke it—to divide these into two principal categories.

The *first category* embraces all those causes where certain specific noxious substances are carried by the blood-current to the kidneys,—substances which irritate these organs, and eventually cause them to become inflamed.

The *second category* comprises those causes which act upon the vessels of the kidneys, and upon the circulation of blood through them, in such a manner as to favor inflammatory changes in these organs; the causes here referred to act chiefly in a mechanical manner.

If we now consider the specific irritants which lead to renal inflammation, certainly those which are admitted by almost everybody to possess this peculiar quality are the poisonous substances already mentioned in the chapter upon Active Hyperæmia, and among which cantharidin occupies the first place as a representative type. The effect produced upon the kidneys by cantharidin will perhaps convey in the clearest manner possible my idea of a specific nephritis. Poisoning by cantharides is certainly rare as a cause of true nephritis; but a thoroughly observed case of it has been quite recently published by Schroff.[1]

I have placed the category of specific causes first in order, because it comprises the larger number of cases.

Among all the specific causes of acute nephritis, *scarlet fever* is the one which is most common. Those who in earlier times reported the occurrence of nephritis in connection with the scarlatinal process were not all aware that the kidney complication was of specific origin, and even at the present day there are persons who hold the view that the renal inflammation that follows scarlet fever is provoked by cold, to which the skin, rendered more sensitive by the disease, is exposed. An unprejudiced observation of the facts teaches us, however, that the frequency of nephritis after scarlet fever varies remarkably in different epidemics, and that it is entirely dependent upon the character of the epidemic. Out of one hundred and eighty cases which were treated by me in an epidemic prevailing during the years 1853 and 1854, twenty-two were succeeded by nephritis; while of eighty-four cases of scarlatina treated by me in the General Clinic in the year 1863, dropsy and nephritis followed in thirteen instances, and with an invariably fatal issue. In other epidemics I have met with scarcely a single instance of this dangerous complication among perhaps a hundred cases. In this connection I wish to call particular attention to the fact that I have very often seen the severest renal affection occurring in the course of convalescence from scarlet fever where the parents have never allowed their children to leave their beds; while neglected children, who could be said to have passed through their attack of the disease absolutely in the streets, have been admitted into the clinical wards and remained entirely free from sequelæ.

---

[1] Ueber Cantharidin und sein Verhältniss zu den spanischen Fliegen. Zeitschrift der k. k. Gesellschaft der Aerzte zu Wien. 1855, S. 480.

In individual instances it is certainly not the intensity of the general or local symptoms of the scarlatinal process which determines the occurrence or the absence of the renal affection. This is often absent in cases of the greatest severity; on the other hand it may lead to a fatal issue after a perfectly mild scarlatinal attack. Still, as a general statement, nephritis is more common in those epidemics which have been called malignant, by reason of the severity of the febrile symptoms and the dangerous complications of all kinds (especially those of a diphtheritic nature) that attend the individual cases.

Care, however, must be taken not to mistake the effect of the febrile action, in its production of albuminuria, for the specific influence of the scarlatina process upon the kidneys. Febrile albuminuria occurs in the severe form of scarlet fever, just as it does in every other form of grave febrile disturbance; but then it coincides with the height of the fever and subsides with the fall of the temperature. The nephritis does not manifest itself in the course of scarlet fever until after a period of almost complete remission from all febrile action, of from one to two weeks' duration, has elapsed, and the eruption has quite disappeared.

In the course of the epidemic of 1853 and 1854—already mentioned above—I had leisure enough every day to examine the urines of very many of my scarlet-fever patients, and I discovered at that time that in the cases where there was severe fever, albumen usually appeared in the urine at the height of the exanthem, disappearing again with the subsidence of the fever. A febrile albuminuria of this kind, lasting for a few days, does not by any means imply that a nephritis will follow. The albuminuria, which signifies the existence of a renal inflammation, appears at a much later date. Out of twenty-two cases in which nephritis occurred in that epidemic, the earliest date of its occurrence was the sixteenth day from the first appearance of the rash, and the latest was the thirty-first day. If an average be taken, the twentieth day will be found to be the mean limit at which this symptom first manifests itself; and it so happens that in relatively the largest number of these cases the albuminuria first showed itself upon the twentieth day. In a subsequent epidemic I was able, in one instance, to make out the commencement of nephritis as early as upon the tenth day of the scarlatinal disease.

Some writers have asserted that every case of scarlet fever is attended by an affection of the kidneys. Scarlet fever, they say, provokes in its course a process of desquamation of the renal epithelium with attendant albuminuria just as regularly as it excites an exanthematous rash upon the skin, succeeded by subsequent peeling off of the epidermis. But from my own careful and exact microscopical and chemical examinations of the urine in a really large number of cases of scarlet fever, I am in a position to assert that this condition of things, for which the term *en*anthem [as opposed to *ex*anthem — TRANS.] has been applied, is a piece of pure fiction. In far the larger proportion of all the cases of scarlet fever, albumen certainly never occurs in the urine; and, throughout the whole course of the complaint, one is able to discover neither renal epithelium nor its fragmentary débris in the sediment of the urine.

Still of this I am convinced, that the post-scarlatinal renal process is kindled by some specific influence, and is not the result of simple mechanical disturbance of the blood-current provoked by long-continued fever heat or collateral blood-flux following the impression of cold upon the external skin. I have already stated my reasons for regarding the mechanical explanation of the nephritis as inadmissible; but in my investigation of the urine after scarlet fever I have striven in vain to find a substance that, in its transit through the kidney, like any poisonous irritant diuretic, could act as the provoker of the inflammation. My failure to find such a substance, however, does not shake my conviction that the pathological changes which take place in the skin in scarlatina cannot of themselves be the cause of the subsequent renal inflammation. Far graver alterations of the skin, following a no less acute or febrile course than that attending scarlet fever, are followed by no similar results. Out of more than three hundred cases of small-pox, which were treated in my Clinic during the last epidemic, there was not a single instance of nephritis. When renal inflammation follows a skin affection, like an extensive burn, for instance, the nephritis is provoked at the height of the malady, not after the subsidence of the pathological process in the skin, as is the case after scarlet fever.[1]

If the conclusions, which Oertel believes he is justified in drawing from his microscopic investigations of the kidneys of persons who have died of diphtheria, should be confirmed by further observations, it may be hoped that the future will throw some light, too, upon the mode of origin of scarlatinal nephritis. That scarlet fever, like all the other acute infectious diseases, is produced by a contagium vivum, and that the reproduction of this living poison, which has found its way into the human organism, must be the cause of the tissue changes which accompany the morbid processes excited by the invasion, is a doctrine which almost everybody at the present day will recognize as well established.

---

[1] I have, at this moment, under my care at St. Bartholomew's Hospital, a case of psoriasis. The man, aged thirty-five, has suffered with repeated returns of his inveterate skin disease at almost every spring and fall of the year since childhood. His attack this autumn, September, 1876, was exceptionally severe. It was attended by a sharp attack of acute parenchymatous nephritis of very typical kind, with moderate dropsy. Both affections, psoriasis and nephritis, are now, on October 21, in process of subsidence. This case illustrates our author's dictum (that the incidental nephritis occurs at the height of the malady) so entirely that the publication of the fact in a note appears to me desirable.—TRANS.

Diphtheria also, like scarlet fever, entails acute inflammation of the kidneys. Oertel[1] has undertaken to show that the special cause of the diphtheritic nephritis is the penetration of micrococci into the kidneys, and their multiplication within these organs. It is perhaps more usual in diphtheria than in scarlet fever, for a slight albuminuria, unattended by any decided change in the kidneys, to take place; indeed, after the patient's death, even a minute microscopical examination may fail to discover any noticeable alteration of the kidneys from their normal state; and in the cases that get well this albuminuria very quickly disappears again. At the same time I am inclined to attribute these cases of albuminuria, too, to some specific influence, since they admit of explanation neither upon the supposition of an elevated febrile temperature nor upon that of a passive hyperæmia; for this albuminuria, which only lasts a short time, often occurs in even very mild forms of diphtheria, which give rise neither to a high temperature, nor to any disturbance of respiration whatever. Diphtheria, too, like scarlatina, does not excite a renal affection in every instance. In several fatal cases of diphtheria affecting the air-passages, I have, from first to last, failed to discover albumen in the urine, and even after death I could discover no evidences of renal disease. The severity also of the renal affection stands in no direct relation to the extent of mucous membrane involved in the diphtheritic process, or to the intensity of the general disease. Even in the cases where, without implication of the respiratory passages, the end has been reached under symptoms of most serious collapse, one may find the kidneys, so far as the naked eye can discover, intact. Upon the other hand, a very slight diphtheria of the soft palate, which has run its course without fever, may terminate in a nephritis with a fatal issue. Generally speaking, however, I am able to confirm the conclusion reached by Oertel, namely, that the kidneys are found most intensely diseased in those cases of diphtheria in which the symptoms of general poisoning have been most prominent. At the same time, I must not conceal the fact that the results of a series of observations and experiments instituted here by Dr. Babbe, and conducted under the eyes of my colleague (Heller), have failed to confirm the views of Oertel regarding the production of the diphtheritic nephritis by emboli composed of bacteria.

---

[1] Experimentelle Untersuchungen über Diphtherie. Deutsches Archiv für klin. Med. 1871, Bd. 8, S. 242; also the article on Diphtheria in the first volume of this Cyclopædia.

The other acute exanthemata—measles, rubeola (Roetheln), small-pox—give rise to acute nephritis far less frequently than does either diphtheria or scarlet fever. If a different view has been expressed by some writers regarding the connection between acute nephritis and small-pox, it is safe to assume that this view owes its origin to the fact that in the hemorrhagic form of small-pox hæmaturia occurs quite often; it is caused, however, not by an inflammation of the kidney, but by hemorrhagic infiltration of the mucous membrane lining the pelvis of this organ. I am willing to state positively that the transitory albuminuria so frequently observed in our last epidemic of small-pox most certainly could not in every case be explained as merely a symptom of the fever; it was due, perhaps, to some specific changes in the renal vessels. However, no macroscopic evidence of renal changes was discovered in the forty-three bodies of small-pox patients on which post-mortem examinations were held during this epidemic, with the exception of two cases, in which the kidneys were the seat of chronic disease. When acute nephritis does arise in the course of measles, rubeola, or small-pox, it probably originates in the same way as the nephritis of scarlet fever and diphtheria.

The acute nephritis which Ponfick[1] found, almost without exception, at the autopsies of relapsing fever patients, may be ascribed to the action of a specific irritant conveyed to the kidneys through the blood, and analogous to that which I have endeavored to demonstrate as the cause of the nephritis in scarlet fever and diphtheria, with all the more certainty, since the fact that relapsing fever is due to the introduction of bacteria may be considered as settled beyond room for doubt. These organisms circulate in the blood, and are carried to the kidneys, where their presence and movements excite inflammation in the same way that the micrococci, according to Oertel, excite nephritis in diphtheria. We should, perhaps, number yellow fever also among the acute infectious diseases that may occasion specific inflammation of the kidneys.

---

[1] Anatomische Veränderungen der Nieren bei Typhus recurrens. *Virchow's* Archiv. Bd. 60. S. 160.

The acute nephritis which in rare cases complicates abdominal typhus is, in my opinion, due not to any specific irritant, but, like the pneumonia, which is not a very rare complication of typhoid, to the alterations in the vessels consequent upon the long-continued high temperature. My reasons for this opinion will be mentioned presently.

Certain local affections of the skin and the cellular tissue, such as erysipelas, carbuncles, and phlegmons, also furnish specific causes of acute inflammation of the kidneys. I do not mean, as was formerly believed, that, in consequence of these cutaneous affections, some irritating substance is retained in the system which would otherwise have been eliminated in its natural way, but that, as the result of these processes, something is bred or originated at the diseased part which obtains access to the circulating fluids, and is conveyed by the blood to the kidneys, there to act as the exciter of inflammation.

The idea that there is a substance which begets inflammation (phlogogenic), as well as one which causes fever (pyrogenic), has long met with favor, and to the former the local extension of the erysipelatous process, for instance, has been attributed. Why should this substance not retain and exert its phlogogenic influence when carried to distant parts of the body? In these cases also positive observations have been recorded, which seem calculated to place the above indicated mode of production of the acute nephritis that complicates certain cutaneous affections upon a surer foundation than mere speculation. Fischer,[1] of Breslau, in a work on the nephritis in carbunculosis, showed that butyrate of soda, a product of the acid fermentation of pus, which is also formed in the carbuncular foci, excites nephritis in animals when it is injected into their veins.

---

[1] I am obliged to quote here from memory, for *Fischer's* book, which I read years ago, is not now at hand.

Moreover, profuse suppuration in other than the subcutaneous strata of cellular tissue may also give rise to acute inflammation of the kidney as soon as, in consequence of the admission of the atmospheric air, decomposition of the pus begins. In the year 1872 I met with two cases of peripleuritic abscess, one shortly after the other, in both of which acute nephritis followed the establishment of an external opening. In the first case the renal inflammation continued until the patient's death, led to excessive dropsy, and was confirmed by post-mortem examination. In the other instance the nephritis was cured before the abscess had completely closed. I lay special stress upon the circumstance that the pus, which, in consequence of the irregular shape of the sac of a peripleuritic abscess, cannot drain freely away, possessed for several weeks, especially in the first of these cases, an intensely fetid smell.

In fact, I have never known acute nephritis to complicate even very extensive purulent collections so long as the absorption of the products of decomposition was prevented by securing a free exit for the pus, or so long as the pus remained in closed cavities within the body, protected from the air. In the latter case the formation of the products of decomposition is restrained.

Finally, perhaps, I may class with the nephritis due to the absorption of the products of decomposition those rare cases which occur in the course of dysentery. Of these I have met with but very few.

Let us now pass to the second category of the causes which, as experience has taught us, may excite acute nephritis. I have already described these causes to be such as act mechanically upon the vessels, and thus affect the circulation of the blood through them. In the preceding section of this work I named cholera as the representative of this category, and explained in detail the manner in which cholera nephritis is produced. In this class of causes I also include catching cold, which, it is true, must frequently be offered as an etiological scapegoat, but the importance of which as a cause of inflammation of the kidneys cannot be doubted. Of the cases of acute nephritis that have come under my own observation, I have been able to refer not an inconsiderable number, with positiveness, to catching cold, since the effect followed exposure to cold almost immediately. One patient got drunk after spending a winter night in dancing, threw himself, half-undressed, on his bed by an open window, and went to sleep. When he woke up his limbs were stiff with cold. From that hour he felt sick, and when I first saw him, a few weeks later, his whole body was dropsical, and his urine was strongly stained with blood. Another patient, while perspiring freely from severe work, left his smithy and went into the open air, with no covering on his shoulders except a shirt, and, while cooling a piece of red-hot iron in water, allowed himself to be wet through with an icy rain. Two weeks later he was brought into the hospital suffering from dropsy and hæmaturia. A third, immediately before his illness set in, broke through the ice while skating, and only succeeded in extricating himself from his icy bath after prolonged exertions, etc.

Whatever views may be held as to the way in which inflammatory affections are caused by exposure to cold, there is one point in which they all agree, and that is that the violent contraction of the cutaneous vessels produced by extreme cooling of the skin, must drive the blood into the internal parts of the body, and consequently lead to elevation of the blood-pressure in the vessels of the internal organs. That this hypothesis applies especially to the kidneys was well demonstrated by Koloman Mueller, in his address, "On the Influence of the Cutaneous Activity on the Secretion of Urine,"[1] before the Medical Section of the Association of German Naturalists and Physicians, at Wiesbaden, in 1873.

Now, whether this collateral fluxion, either alone or in connection with alterations in the temperature of the blood, is the agent which, after a sudden cooling off of the overheated skin, excites the development of inflammation of the tonsils in one person, of a catarrhal inflammation of some mucous tract in another, and finally of an acute nephritis in a third, cannot as yet be decided with certainty. Dr. Gustav Wollner, in an inaugural dissertation at Erlangen, describes some cases of acute nephritis which followed very energetic employment of the inunction cure for scabies. It was not quite clear whether the nephritis in these cases was due to catching cold after the repeated hot baths, or to the dermatitis caused by the inunctions.

---

[1] Published in the Experimentelle Pathologie und Pharmakologie.

The inflammation of the kidneys and of other organs consecutive to extensive burns of the surface of the body, is unquestionably produced in the same way as the inflammation which is due to catching cold. It is well known that the destruction of the skin produced in this way, when extensive, causes a general depression of the temperature of the body in consequence of the great loss of heat; it acts in the same way as a continued abstraction of heat by cooling of the uninjured skin. Falk's[1] opinion that the inflammation of internal organs following extensive burns is due to the destruction of the red blood-corpuscles which were present in the vessels at the site of the injury, and were thus exposed to the excessive heat, is not supported by a sufficient number of positive facts; he believes that the morphotic fragments of the destroyed blood-cells, together with the chemical products of their decomposition, are swept with the current of the blood into the affected organs and there excite inflammation. Falk's favorite comparison of the effect of an extensive burn with the effects of poisoning by carbonic oxide gas, which also occasionally produces acute nephritis, does not appear to me by any means to support his views as to the nature of the process. Carbonic oxide gas, according to my own observations, induces a general paralysis of the blood-vessels by preventing the oxidation of the blood. As a consequence of this paralysis, inflammation, which terminates in the formation of blebs, occurs at all those points of the skin which are exposed to constant pressure or to other mechanical injuries. This inflammation can be prevented by carefully protecting the skin from all hard pressure, just as we prevent its occurrence in limbs whose vaso-motor nerves are paralyzed from any other cause. I always found the contents of the vesicles that form on the skin after poisoning by carbonic oxide to be clear and serous, and never tinged by the blood-pigment.

---

[1] Ueber einige Allgemeinerscheinungen nach umfangreichen Hautverbrennungen. *Virchow's* Archiv, Bd. 53, S. 27.

I also attribute to the mechanical disturbance of the circulation those cases of acute renal inflammation which occasionally develop in the course of abdominal typhus, and of other diseases attended by an unbrokenly continuous high temperature. I have already stated, while discussing the subject of albuminuria, that it is a pretty constant symptom when there is persistent high fever, and that I attribute it to the dilatation of the vessels, constantly associated with elevation of temperature, as well as to other alterations in the walls of the vessels. I also stated that I believe this condition of the vessels, when long-continued, to be capable of inducing serious disturbances in the nutrition of the kidneys, and must consequently admit the possibility of the occurrence of an actual inflammation. In point of fact, however, this does not often occur. Out of about one thousand carefully studied cases of typhoid fever which have come under my care, either in my hospital or private practice, in the course of many years, I have only had to record the complication with true nephritis in two cases. By an odd coincidence both these cases came under observation at the same time, and just when I was preparing this article. Only one of them terminated fatally ; it is the only case in which I have found at the autopsy recent nephritis in addition to the typhoid lesions in the intestines. My experience tallies closely enough with Buhl's[1] observations ; out of three hundred fatal cases of typhoid, he only once or twice met with general dropsy dependent on recent renal changes. My second case of nephritis complicating typhoid terminated in perfect recovery. I once saw acute nephritis arise in the person of an officer in the course of a *genuine pneumonia*. The renal affection produced extreme general dropsy, but after the expiration of two months complete and permanent recovery ensued.

The way and manner in which acute nephritis originates in acute rheumatism is a question on which I have been able to form no opinion. I have repeatedly observed the commencement of a hemorrhagic nephritis in the course of acute articular rheumatism, so that I have been compelled to admit that under certain circumstances the rheumatic process may be the exciting cause of this disease of the kidneys. Johnson,[1] too, describes a case of rapidly fatal nephritis, which had developed during an attack of rheumatic fever. I have found no other cases reported in the literature of the subject; Rayer's néphrite rhumatismale, and néphrite albumineuse in connection with rheumatism, refer to very different affections; under the first appellation the Parisian pathologist describes the embolic processes in the kidneys dependent on heart disease, and in the second he has in view merely the occurrence of painful articular affections in the course of a chronic affection of the kidneys, which he believes to be rheumatic in character.

---

[1] Lungenentzündung, Tuberculose und Schwindsucht. Zwölf Briefe an einen Freund. München, 1872, p. 47.

It is true that I have not had an opportunity to perform an autopsy on any of the cases of nephritis under my care, in which the affection appeared during an attack of rheumarthritis acuta (all the patients are still living). Still the following description of one of these cases will, I think, show that my diagnosis was correct, and that I have not made the mistake of confounding a hemorrhagic infarction of the kidney with diffuse renal inflammation.

Case XII.—S. G., a strong, well nourished country girl, aged twenty-one years, was admitted into our hospital on December 2, 1873. Seven years before she had been confined to her bed for some months with an attack of acute articular rheumatism. Three months ago she had suffered for some weeks from rheumatic pains, but since that time had been entirely well. Four days previous to her admission she was seized with pains at first in both ankles, and then in both knee-joints, accompanied by fever and palpitation of the heart.

Condition on admission: Both wrists swollen and painful; all the other joints free. The area of præcordial dullness increased in all directions beyond its normal limits. The apex beat can be felt in the line of the nipple in the interspace between the fourth and fifth ribs. Systolic murmur audible at the apex, with a muffled diastolic sound. Pericardial friction sound over the large vessels at the base; heart sounds indistinct. Pulse 80, of moderate volume. Evening temperature 40° C. (104° Fahr.). A trace of albumen in the urine. During the following days the fever continued high, and the pericardium became considerably distended with exudation, but the articular affection subsided, and the albumen disappeared entirely from the urine. About the middle of December the pericardial exudation was reabsorbed, but the patient had repeated and severe fainting-fits, which necessitated the employment of stimulants. The fever remained high.

---

[1] l. c., p. 276.

On the 28th of December she complained of sharp, stabbing pains in the right side, with pleuritic friction sounds in the same situation. The symptoms of pleurisy on the right side continued until the middle of the month of January, 1874. From the 9th of January the fever gradually diminished, and after the 12th even the evening temperature did not rise above 38° C. (100.4° Fahr.).

On the 17th of January the evening temperature suddenly ran up to above 40° C. (104° Fahr.), and the pulse to 120; there was dyspnœa, without perceptible changes in the respiratory organs. The high febrile temperature continued for several days, while a systolic murmur, which became louder from day to day, was heard over the origin of the aorta, and was transmitted into the arteries of the neck, indicating the occurrence of endocarditis of the semilunar valves of the aorta. At the apex a loud diastolic murmur was now heard in addition to the systolic murmur previously noticed.

Towards the end of January the fever moderated, and no longer exceeded 39° C. (102.2° F.) in the evening; the pulse, however, was still frequent—104—but fell to 90 in the course of the first week of February. On the 8th of February there was some swelling of the left hand and elbow-joint, with a moderate elevation of temperature—39.2° C. (102.6° Fahr.). This swelling continued for an entire week, with constant though moderate febrile movement.

During the course of this week the bloated aspect of the patient attracted attention, and a more careful examination of the urinary secretion was instituted. The urine, however, contained no trace of albumen, but only a small quantity was passed; on February 14th, for example, only 300 c.c.

On February 15th the patient passed 500 c.c. of dark colored but clear urine, sp. gr. 1016, containing traces of albumen; about the middle of the same day she had a violent rigor, followed by an elevation of temperature up to 41.2° C. (106.2° Fahr.). Pulse 128. Resp. 44. Towards evening the temperature fell spontaneously to 37.8° C. (100° Fahr.), and the pulse to 104. During this violent access of fever the patient retched a good deal, and repeatedly vomited quantities of bitter yellow mucus, and complained besides of dull pains in the back and præcordial region.

On the morning of February 16th 500 c.c. of very dark colored urine, of 1014 sp. gr., were passed. The entire quantity of urine was clouded by urates. At first the fluid cleared on heating, but when the application of heat was continued it coagulated into a stiff jelly. In the sediment I found many white blood-cells, a few red ones, small, perfectly hyaline casts, and epithelial *débris*. The quantity of blood contained in the urine increased daily. On February 18th 500 c.c. were passed, of a dark, blackish-red color, sp. gr. 1014, giving a thick sediment of blood-cells, blood casts, and scanty hyaline casts. The filtered urine contained 1.08 per cent.

of albumen = 5.4 grammes; urea, 2.1 per cent. = 10.5 gr.; chlorides, 0.8 per cent. = 4.0 gr.

On the 24th of February the percentage of albumen in the urine (quantity passed in 24 hours 850 c.c., sp. gr. 1016) increased to 1.525 per cent. = 12.962 gr. From this date the quantity of albumen and of blood gradually diminished. The quantity of urine passed, however, remained scanty, and in consequence of this the dropsy, which had been apparent even before the hæmaturia commenced, increased until the entire body was enormously swollen. The articular affection which had previously existed disappeared, but fever of moderate intensity continued.

About the middle of March diarrhœa set in, with violent pains in the body, the stools being often bloody. An excessive elevation of the temperature again occurred. On March 31st it reached 41.2° C. (106.2° Fahr.). During this time the constantly frequent pulse preserved a moderate tension.

From the beginning of the month of April the diarrhœa disappeared, the fever gradually subsided, and the previously scanty urinary secretion increased in quantity. Still, the month of May had set in before the urine entirely lost its bloody color, and the quantity passed reached the constant daily average of 1,200 c.c., and the dropsy disappeared.

On the 14th of June the patient was discharged from the hospital at her own request. During the last few days of her stay in the hospital her urine still contained traces of albumen; about 1,200 c.c. were passed daily, sp. gr. 1015. A few hyaline casts, some red blood-cells, and a considerable quantity of renal epithelium were found in the sediment.

On the 19th of December, 1873, the patient presented herself again at the clinic. She felt well, but looked pale, and her face was a little puffy. There were lesions of the mitral and aortic valves. The urine was very pale, sp. gr. 1004; it contained traces of albumen, and a few white blood-cells were found in the sediment; no casts could be discovered.

The results of the examinations of the urine and the whole course of the renal affection prove that I had to deal with a true nephritis in this case. The changes revealed by the examination of the urine, undertaken six months after the discharge of the patient from the hospital, would seem to indicate that the nephritis had resulted in atrophy of the kidney. I am all the more inclined to believe this, because a previous case of nephritis complicating acute articular rheumatism, which came under my observation, had terminated in the same way. In a third case, which was observed in my clinic during this winter (1874 and '75), recovery from the renal affection took place after a few weeks, although the articular malady had not entirely subsided.

It is an important point, or at least one which must not be overlooked, that, in all the three cases in which I saw acute nephritis set in as a complication of acute articular rheumatism, the patients were suffering from recent rheumatic endocarditis at the time the renal affection began. This fact arouses the suspicion that some products of inflammation from the left side of the heart might have been carried into the renal vessels, and been the cause of the nephritis, which, in all three cases, set in with great violence. I can, however, positively assert one thing, and that is that no large hemorrhagic infarctions were developed in any of these cases. That is evident from the entire course of the disease.

I have still to speak of a special form of nephritis, which is peculiar not only as regards its cause, but also as regards its course. I refer to the renal inflammation which is so often developed during pregnancy. On account of its peculiar character I will describe this form of nephritis separately in an appendix to this chapter. I must also state that acute hemorrhagic inflammations not unfrequently set in where chronic renal disease already exists. I have observed such acute attacks in the course of *Chronic Parenchymatous Nephritis* and when the kidneys were in a state of *Granular Atrophy*. Whether this complication has also been observed in cases of amyloid degeneration of the renal vessels, I am unable to say. Hemorrhagic or acute diffuse nephritis occurs relatively very frequently during childhood, because the causes that give rise to it (scarlatina and diphtheria) are more common among children than among adults.

Pathology.

From what has been stated with regard to the etiology, it follows that acute diffuse nephritis may owe its origin to a great variety of causes, and that its course must be correspondingly variable. While laying down this proposition, I must, it is true, admit that the affection which I describe as acute diffuse nephritis comprises perhaps a number of different processes;

however, neither pathological anatomy nor clinical observation has furnished us as yet with the means of differentiating these various processes. Anatomically, as well as symptomatically, the manifestations of the disease differ merely in degree. The dissimilitude is limited solely to the genesis. No matter how the individual cases may originate, the anatomical changes in the kidneys and the clinical symptoms are as liable to be slight as severe. The primary disease does, in a few cases, exert a slight influence upon the grade, the duration, and the issue of the malady. Cohnheim, in his experiments on inflammation, succeeded in producing essentially the same changes in the affected vessels and tissues by the employment of different modes of irritation. At the same time, I do not by any means wish to deny that the type of the affection, when it follows some one particular exciting cause, is, as a rule, more severe, while, on the contrary, when it is due to some other particular cause, it is, as a rule, mild; that, for instance, the duration of the acute nephritis which results from a severe cold is more prolonged, and its detrimental influence on the general health is more intense, than in the majority of the cases of scarlatinal nephritis. It is certain, however, that we do not know beforehand how intensely the physiological properties of the walls of the vessels may be affected by the action of any one cause; or, in other words, whether the quantitative effect stands in any precise relation to the quality of one of the many causes of acute parenchymatous nephritis enumerated above.

General Summary of Course and Symptoms.

In many cases of acute nephritis the clinical history and the course are materially influenced by symptoms dependent upon the primary disease; and the symptoms will also vary greatly, according to the severity of the renal disease.

The commencement of the renal changes may be attended in one case by symptoms of high fever, while in another there is no fever at all. In some rare instances it is marked by violent subjective disturbances, but far more frequently there are no abnormal sensations that attract the attention of the patient. The first effects of these changes are seen in certain functional disorders, consequent not only upon the diminution in the quantity of urine which is secreted—which sometimes amounts to complete suppression—but also to essential changes in its chemical composition by the addition of albumen and usually also of blood.

The cases which set in violently with active fever are preeminently those in which at the commencement of the disease frequent and serious—indeed, often alarming retching and vomiting, are observed. Anorexia and dyspeptic symptoms are present in most of the severer cases. Extreme anæmia and great loss of strength are developed in a very short time, even in the cases unattended by fever. Very frequently, however, these symptoms are present before the renal disease sets in, being dependent upon the primary disease. In very mild cases all evidences of the disturbance of the renal functions may disappear in the course of a week or two. These mild cases of nephritis are not uncommon after scarlet fever, are the ordinary rule after diphtheria, and are also frequently met with in connection with other exciting causes. The renal inflammation may then be regarded as cured ; even in such mild cases, however, relapses occasionally occur. On the other hand, in the more severe cases, where the irritation that excited the inflammation has caused a more profound alteration of the physiological condition of the renal vessels, and thus produced more intense disturbances in the nutrition and the functions of the kidneys, recovery never sets in so rapidly ; but, on the contrary, the affection very often terminates fatally. The nephritis which is due to catching cold presents, as a rule, this more violent type, as do also many cases of nephritis consecutive to scarlet fever and various other exciting causes.

I have found that, with the exception of a few instances of choleraic nephritis, every case of acute parenchymatous nephritis in which complete suppression of the urinary secretion took place terminated fatally, even when small quantities of bloody urine were again passed after prolonged suppression. In such cases the excretion of urine never again became normal in quantity. In these very acute cases death took place after the patients had become more or less dropsical, usually during uræmic convulsions, or in the coma that succeeded them, more rarely with the symptoms of general collapse without any antecedent convulsions. When a case of acute parenchymatous nephritis follows a slow course, it leads in the majority of cases to dropsy, which is sometimes slight and sometimes excessive, and which may last for months, subsiding and reappearing at intervals. Not uncommonly a commencing puffiness of the face or an œdema of the ankles is the first symptom that attracts the attention of the patient and causes him to seek medical advice. The subjective symptoms which the renal disease causes may be so slight as to attract no attention, or may even be altogether absent.

The dropsy itself may be the immediate cause of death, either in consequence of the accumulation of excessive quantities of fluid in the serous cavities of the body, or by acute œdema of a large portion of the pulmonary tissue, or in rare instances by acute œdema of the duplicatures of mucous membrane at the entrance into the larynx.

Lastly, among the causes of a fatal termination in acute parenchymatous nephritis, I must also mention suppurative inflammation of the serous membranes—a rather frequent occurrence, especially in the pleuræ and the pericardium, and even in the pia mater of the brain. However, the great majority of all the cases of acute parenchymatous nephritis terminate in recovery—the milder cases, indeed, as has been already mentioned, within a short period; a great many even of the severe cases which have occasioned considerable dropsical swelling also recover. With regard to the length of time that elapses before the disease terminates in recovery, there are variations in different cases which evidently depend on the nature of the exciting cause of the renal affection. Whereas a scarlatinal nephritis that lasted two months would be considered exceptionally long, an acute nephritis due to a severe cold is apt to be much more protracted, and sometimes continues more than six months.

The rarest termination of acute parenchymatous nephritis is unquestionably its conversion into a chronic renal affection that continues for years. I must repeat here what I have already insisted on: that the opinion that the process I am now describing constitutes the regular prelude to those chronic renal affections which will be described in the following chapters is erroneous, and exerts an injurious influence on practice.

The cases of acute nephritis that originate from cold are the most liable of all to develop into a chronic affection; the cases which complicate acute articular rheumatism may also terminate in this way, but I have never known the nephritis of diphtheria to assume a chronic form. Among all the cases of scarlatinal nephritis, numbering certainly not less than fifty, which have come under my observation, there was only one which lasted eighteen months, and in this case recovery finally took place; of the rest, the longest duration was three months in one case, while a third and a fourth lasted two months. All the remaining cases either died or got well after a very short illness.

Summary of the Post-mortem Appearances.

The bodies of persons who die in the course of acute parenchymatous nephritis present, in addition to the renal changes and their results, often exceedingly varied pathological changes in other organs, corresponding to the various causes which may occasion the inflammation of the kidneys. I shall not take into consideration here the lesions which appertain to the primary disease.

When the renal affection itself has been the essential cause of death, the body is, as a rule, more or less dropsical. The dropsy almost always affects by preference the subcutaneous areolar tissue; less frequently there is considerable effusion into the pleura, the pericardium, or the peritoneal cavity; still less frequently extensive pulmonary œdema; and the rarest form of all is marked œdema of the mucous membranes—for instance, of the folds at the upper part of the larynx. In many cases the immediate cause of death is a simple sero-fibrinous exudation into one pleural sac, or into the pericardium, or a purulent meningitis, or an inflammatory infiltration of a large portion of the lungs.

The essential lesions are of course to be found in the kidneys. These organs are found in various stages of change, corresponding to the duration and intensity of the inflammatory process; the two organs are without exception affected to an equal degree.

In the severer grades of the affection the kidneys are enlarged in toto, and swollen so that the organ may reach twice its normal weight (Ponfick). The capsule of the kidney is tightly stretched, so that it gapes distinctly as soon as it is incised. The increase in bulk affects the cortical substance especially. The consistence of the acutely inflamed kidney is diminished; it has a doughy feel, and the tissue of the cortex is softer and more easily broken down than in the normal condition. The color of the swollen cortical substance on section is rather paler than normal; it has a dull, grayish red aspect.

Very frequently dark red points (glomeruli distended with blood) and streaks stand out in contrast to the paler ground, and even with the naked eye small punctiform extravasations on the surface may be distinctly seen.

Bright describes a case of this sort in his Reports of Medical Cases. In other cases the hyperæmia of the cortical substance of the kidney is less marked. The pyramids, on the contrary, are always greatly congested, sometimes bluish red in color; and if the general swelling is intense, the contrast between the bright red medullary substance and the pale gray cortex is very marked.

In still other cases, finally, the cortical substance, in consequence of general hyperæmia, assumes a dark, dirty, grayish red color; in two cases of scarlatinal nephritis in children I found the kidneys presenting this appearance. I remember well that I was impressed with the fact that the swelling of the scarlatinal kidneys had caused principally an increase in thickness to such an extent that the natural flattened shape of the organs from before backwards was almost entirely lost, and they had become very nearly cylindrical in shape.

However, the kidneys do not present the above-mentioned signs of extreme hyperæmia in every case of scarlatinal nephritis that terminates fatally. On the contrary, it more frequently happens that no very essential deviation from the normal appearance of the kidney, with the exception of the swelling of the cortical substance, can be discovered by the naked eye; the cortex is more apt to present a pale than a hyperæmic tint. This evidently depends, in some measure, upon the length of time that the disease had existed before death.

I found the more intense degrees of hyperæmia in those cases in which death had been ushered in by uræmic symptoms shortly after complete suppression of the urinary secretion; whereas the swollen kidneys presented a paler aspect when the disease had run a protracted course, and death had finally been caused by dropsy or by a complication with inflammatory affections of other organs. In such cases the pale reddish gray cortical substance often presents a mottled aspect, caused by scattered lines and streaks of a distinctly yellowish color, which stand out in sharp contrast to the surrounding tissue. In those cases in which the patient does not die of the renal disease, but of some affection totally independent of it, such as unhealthy suppuration in the areolar tissue of the neck or some other sequel of scarlet fever, the swelling of the diseased kidneys is sometimes very slight in degree.

Between this slight, scarcely perceptible swelling and cloudiness of the kidneys, and those extreme changes which I have just described, we meet at the post-mortem table with every possible intervening stage. We have to deal here simply with differences in degree, which correspond to the differences in the intensity of the functional disturbances observed at the bedside previous to death; these may vary from temporary albuminuria of short duration, or slight hæmaturia, to complete and permanent suppression of urine. We certainly cannot agree with the opinion expressed by some writers, who, misled by the different appearances presented by the kidneys in different cases, and by the different degrees of disturbance of function during life, have endeavored to establish an essential distinction between the mild and the severe cases of acute parenchymatous nephritis, and separate them from one another as specifically distinct processes under the name of catarrhal and croupous nephritis.

The result of this effort is that one writer describes scarlatinal nephritis as a catarrhal, another as a croupous, and a third as an interstitial inflammation, probably because the first based his description of the affection upon the results of the anatomical examination of kidneys that had undergone but slight changes, while the others drew their inferences from the lesions in organs that were more or less severely affected. Unquestionably the kidneys of an individual who died of scarlet fever or diphtheria, and whose urine for a few days before his death merely contained albumen, a few casts and a little blood, will not present the same appearances as the kidneys of another person who has succumbed to the effects of the same malady, but who for days before his decease had not secreted a drop of urine, whose body was swollen beyond recognition in consequence of the obstruction to the excretion of water through the kidneys, and in whom death took place during coma or uræmic convulsions. Nevertheless, the causes and the nature of the renal changes, albeit so different in degree, are in both instances the same. Any one who has seen many cases of scarlet fever, and has had abundant opportunities, which he has carefully improved, of investigating the disease anatomically, will agree with my statement that in scarlet fever, not only clinically, but anatomically, the inflammatory affections of the kidney of every possible grade, from the slightest to the most severe, are met with, and he will feel bound to dissent from any such classification of the disease as the one mentioned above.

Even the microscopical appearances in the diseased kidneys do not, by any means, furnish sufficient evidence in favor of such a differentiation. We simply learn from them that the intensity of the structural changes may vary greatly in the different cases, but that these changes, with the exception of the organic substances that have been carried into the renal substance with the blood, and have constituted the special exciters of the inflammation, are identical in nature. They affect the epithelium and the interstitial tissue. The only question that is still open to dispute is as to which of the two parts of the renal tissue is first affected; and it is disputed especially because it involves the question whether the changes in the epithelium are to be ascribed to an active process, or are to be regarded as the expression of an anæmic necrosis which attacks the cells, because the swelling of the interstitial connective tissue compresses the blood-vessels and cuts off the nutrient supply—in one word, the question at issue is: Is there really a parenchymatous inflammation of the kidney?

Let us, however, consider first the objective appearances. Even in the very mildest cases, where death is due, not to the nephritis, but to some other organic affection, we never fail to find swelling and cloudiness of the epithelial cells due to the deposit of granular masses in them; whereas in these mild cases there are no clearly evident changes in the interstitial tissue. On the other hand, the changes in the interstitial tissue are never absent in the higher grades of general renal swelling. The interstices between the renal tubules, which are distended and dilated by the swelling of their epithelial lining, also appear wider (swollen), and contain lymphoid elements in varying numbers. The longer this condition of renal swelling continues, the more advanced is the next change that takes place in the swollen epithelium; drops of fat appear in the cellular stroma in constantly increasing numbers. These changes in the epithelium and the interstitial tissue are often almost entirely confined to the cortical substance, the straight tubes of the pyramids presenting scarcely any perceptible alteration. Moreover, even in the cortical substance all parts are not affected to an equal degree; renal tubes whose epithelial linings are but little altered are not unfrequently found in close proximity to very fatty portions, the epithelial cells of which have partly broken down into a fatty detritus. The yellow patches which are found in the more protracted cases, and which stand out in such plain contrast to the pale gray or darkly hyperæmic tissue surrounding them, are probably due to such a fatty degeneration. The only essential and constant microscopical appearances in acute parenchymatous nephritis are the changes in the epithelium of the tubules that have just been described. For these changes, and for the process which induces them, I have no hesitation whatever in retaining the name of *acute parenchymatous inflammation of the kidneys.* In all cases, the mildest as well as the most severe, we have to deal with an infiltration of the epithelial cells—with an albuminous transudation from the blood-vessels. With this process the changes in the intertubular tissues have nothing to do; observation has taught us that the same process takes place to a certain extent even in those mild cases where no change in the interstitial tissues can be discovered. Whether the swelling that subsequently takes place in this interstitial tissue, by compressing the nutrient vessels, promotes this fatty degeneration and destruction of the epithelial cells that were previously in a condition of inflammatory swelling, is another question. Even this, however, I am by no means willing to admit unconditionally. For if we ascribe this fatty degeneration of the inflamed parenchyma unconditionally to an anæmic necrosis, it will be impossible to understand how it is that at the spot where the inflamed and swollen cells are undergoing destruction, the eminently nutritive act of new formation can at the same time be performed and compensate for the destroyed cells. According to the observations of Mayer and Kelsch, however, this unquestionably occurs in cholera nephritis, and it is highly probable that it also takes place in other forms of acute parenchymatous inflammation. For, as has been already stated, the great majority of even the severest cases of this disease, when they do not end fatally, terminate in complete recovery, the cases that are followed by atrophy of the kidneys being exceedingly rare and exceptional.

In the cases that terminate in recovery it is also fair to presume that the white blood-cells which had migrated into the intertubular tissues are again taken up by the lymphatic vessels.

Besides the essential lesions just described, we constantly find, in the microscopical examination of kidneys in a condition of acute inflammation, cylindrical plugs here and there in the lumina of the tubuli. In this affection I have never seen any but perfectly homogeneous, small, and pale casts in the kidney itself. I always found them only in those tubes in which the epithelial lining was still complete, and was but little or not at all altered. Ponfick, speaking of the kidneys in relapsing fever (Recurrensnieren), says that these casts which, like myself, he calls fibrinous, are most frequently situated in the inferior portions of the convoluted tubes and in Henle's loops.

Hemorrhages are usually but not invariably met with in acutely inflamed kidneys. Ponfick has shown that these emanate from the Malpighian tufts; he found effusions of blood often in very considerable quantities in the space between Bowman's capsule and the capillary coils of the glomeruli.

These extravasations into the tubuli uriniferi appear on section of the affected kidney as dark red or brownish streaks. The lumina of such tubes are seen under the microscope to be filled with blood-casts, and blood-casts are also found in the urine.

The renal vessels, as a rule, exhibit no perceptible alterations. I have already stated that the glomeruli are often so greatly distended with blood that they can be seen as red points by the naked eye; in other cases, particularly in the more advanced ones, they are pale. Klebs[1] describes, under the title of "Glomerulo-nephritis," a condition of the glomeruli after scarlet fever, to which he attributes the occasional total suppression of the urinary secretion. He found the glomeruli in fine sections remarkably bloodless; macroscopically, by reflected light, they appeared as white points, and on microscopical examination the entire space within the capsule was found filled with small, somewhat angular nuclei. His further investigations led him to conclude that this multiplication of the nuclei was due to a proliferation of the cells in the interstitial tissue of the glomerulus, by which the vessels of the tuft were compressed. Klebs' own experience, however, is not sufficient to enable him to state positively that similar changes may occur independently of the scarlatinal process. I am not aware of any observations by other authorities that confirm this view; as for myself, I have not had an opportunity of examining the kidneys after scarlet fever since it was published. I have already referred to Oertel's statement, that in the renal affections following diphtheria great numbers of micrococci, and even exuberant proliferations of the same, can be discovered both in the renal tubes and in the Malpighian bodies.

---

[1] Handbuch der pathologischen Anatomie, S. 645 ff.

The investigations instituted by Dr. J. M. Babbe,[1] in the Pathological Institute attached to this University, which is under the direction of my colleague, Prof. Heller, and described by him in an inaugural thesis, have not furnished a confirmation of Oertel's statement. On the other hand, I am indebted to my colleague Heller for the information that he has repeatedly found the blood-vessels (in two cases a great many of them, and sometimes over a considerable area) and their branches in acutely inflamed and swollen kidneys greatly dilated, and plugged with masses which, when low magnifying powers were used, presented a peculiar grayish yellow appearance. With higher powers these masses were found to consist of extremely minute, strongly refracting granular particles of equal size, and placed at equal distances from one another—spherical bacteria. These bacteria-emboli had, it is true, given rise to the formation of foci which were easily distinguishable in the swollen kidneys; nevertheless, it seemed to me that these observations would be of great value in explaining the diffuse inflammation of the kidneys in certain cases. In all of Heller's cases the subjects had died with pyæmic symptoms: one was a *puerperal* woman, with purulent peritonitis, gangrenous patches in the portio vaginalis uteri, an eschar of the bladder resulting from pressure, and ulcerative endocarditis; another was a girl fourteen years of age, with endocarditis of the mitral valve, punctiform deposits in the muscular substance of the heart, punctiform hemorrhages, and spots of encephalitic softening in the brain, and small hemorrhagic infarctions in various other organs, also with diphtheria of the pharynx and larynx, and finally, with fistulous openings into the knee-joint; the last was an individual with a dissecting wound of the left forearm, ecchymoses beneath the endocardium, slight purulent peritonitis, etc.

These cases recalled to my mind a case of hemorrhagic nephritis occurring as a complication of ulcerative endocarditis, which came under my observation in 1863. The patient was a very robust Finn sailor, who stated that previously he had always been healthy, but was seized with a severe fever while travelling; he became extremely dropsical, and succumbed to his malady a few weeks after his admission into my wards.

---

[1] Ueber das Vorkommen von Pilzen bei der Diphtherie. Kiel, 1874.

Analysis of the separate Symptoms.

I have already stated that the symptoms of acute parenchymatous nephritis are in the majority of the cases accompanied by the symptoms of the primary disease. The febrile movement must often be ascribed to the latter, for the renal inflammation in itself does not necessarily excite fever in every case, and does not even at the time of its outbreak always cause an exacerbation of a pre-existing fever. In other cases—and here belong especially those which are due to catching a severe cold, as well as those which complicate endocarditis—I have seen the renal affection commence with severe febrile phenomena, occasionally even with an initial rigor. I have frequently seen the temperature rise above 40° C. (104° F.) in such cases; the fever, however, in uncomplicated cases is always of short duration, and in the further progress of the affection, elevations of temperature only occur upon the outbreak of uræmic convulsions, or when some secondary inflammatory process has been set up in some other organ.

Local symptoms which directly indicate a renal affection are seldom severe in acute parenchymatous nephritis, and are often entirely absent. It is only in rare cases that the patients complain spontaneously of aching pains in the lumbar region, which may radiate into the inguinal region and the inner surface of the thigh. The region of the kidneys is very often tender upon deep pressure, sometimes extremely so.

Once, in an extremely thin woman twenty-five years of age, who, moreover, had never had a child, I was able to map out the two kidneys distinctly by palpation through the relaxed abdominal walls; they had retained their natural form, but were considerably enlarged. This woman had been passing bloody urine for some weeks before this examination was made, and was already very much reduced.

A subjective symptom, which is present in many cases, is calculated to direct the attention of the patient to his urinary organs before any dropsy appears; it is a frequent and excessively urgent desire to micturate, while at each successive attempt to relieve this desire only a few drops, often of bloody urine, are evacuated. This symptom, however, is by no means constant, and is apt to disappear very quickly. The most essential symptoms consist in disturbances in the functional activity of the kidneys, in consequence of which both the quantity and the composition of the urine secreted are altered.

In nearly every case of acute parenchymatous nephritis that I have seen the quantity of urine secreted at the commencement of the affection fell far below the normal average—to a few hundred cubic centimetres in the twenty-four hours; sometimes the secretion was entirely suppressed, and then, as has been already mentioned, death regularly ensued within a few days. In the milder cases the urine usually continued scanty for some time, often for several weeks. On the other hand, when the disease followed a favorable course, the recovery was so often preceded by an abnormal increase in the quantity of urine secreted that this may be regarded as the rule; in many cases 2–3,000 c.c., and even more, were passed daily for weeks. Still, even in cases that recovered, it was not uncommon for considerable fluctuations in the quantity of the urinary secretion to occur, it being alternately increased and diminished. The natural equipoise of the secretion is not permanently re-established until convalescence is complete.

At the commencement of the attack the urine passed is always cloudy, partly in consequence of the separation of urates, partly in consequence of an admixture with morphotic elements, and this cloudiness sometimes continues until recovery. Its color varies, the variations depending partly on the quantity passed, partly on the appearance and disappearance of blood in it. The addition of blood, according to its quantity, gives the urine at one time a pale tinge, like that of water in which meat has been washed (fleischwasserähnliche), and at another a dark blackish red color. When the quantity of blood contained in the urine is very considerable, a very thick sediment of a chocolate brown color is sometimes deposited; this consists of urates and blood-corpuscles, some of which are in a state of perfect preservation and some broken down.

I have always found the reaction of the urine in acute parenchymatous nephritis to be acid. The specific gravity depends principally upon the quantity passed, and consequently varies greatly during the course of the affection; at the commencement, when very little urine is excreted, it is often much higher than normal;[1] at this time I have frequently found it as high as 1031. When the secretion becomes more abundant its specific gravity of course falls, and remains often for many weeks abnormally low—from 1009 to 1011—sinking sometimes to 1006.

The quantity of the solid constituents of the urine, especially the urea, also varies with the specific gravity. Only at the outset of the affection, when the urine excreted is very scanty but dense, do we find by analysis anything like a normal percentage of urea; it is, however, seldom above two and one-half per cent., and as a rule is less. Even more marked than the diminution in the percentage of urea at the commencement of the inflammation is the falling off in the total quantity excreted. This naturally follows from the fact that the actual quantity of urine excreted daily at this period is, as a rule, far below the normal. I have several times found that adults, even when the outbreak of the acute parenchymatous nephritis was attended by fever, excreted only from eight to ten grammes of urea in the twenty-four hours.

If, in the further progress of the affection, the urinary secretion becomes more abundant, the percentage of urea, along with the specific gravity, falls still lower, and is often very much below the normal. I have often found it as low as one per cent. In one case of scarlatinal nephritis, when 1,500 c.c. of urine were passed daily, with a specific gravity of 1006, I found only 0.8 per cent. of urea. It is self-evident that the small percentage of urea in the urine may be counterpoised by an increase in the quantity of the urinary secretion, so that the total amount of urea excreted may not only be equal to, but may exceed, the normal. A robust journeyman mason, for example, in the seventh week after the commencement of a nephritis that was secondary to an extensive phlegmon, passed in one day 3,500 c.c. of urine containing one per cent. of urea; in all, therefore, thirty-five grammes of this substance.

---

[1] I will call attention here to an error which first crept into the earlier British publications on Bright's disease, and held its ground for a long time, and that is that the albuminous urine excreted during the existence of diffuse disease of the kidneys always has a low specific gravity. The opposite is often the case at the commencement of acute parenchymatous nephritis, and is frequently observed for a long time during the course of chronic parenchymatous inflammation.

I have paid little attention to the rest of the normal constituents of the urine in acute parenchymatous nephritis, except the chlorides, of which I may say that in the cases attended by general dropsy, they are excreted in diminished quantities (four to five grammes per diem). In one case of nephritis, secondary to peripleuritis, in which there was very little anasarca, although for weeks the urine contained blood, twelve analyses of the urine undertaken during the six weeks that the disease lasted showed that on an average fourteen grammes of common salt were excreted diurnally by the kidneys.

In every case of acute parenchymatous nephritis that I have seen, the urine has contained albumen, sometimes in small, sometimes in pretty considerable quantity. In no case, however, have I known the percentage of albumen in the urine to attain the enormous height which it not unfrequently attains in the course of chronic parenchymatous nephritis. The largest quantity of albumen that I have observed occurred in the case of nephritis complicating acute articular rheumatism detailed above; it reached 1.525 per cent., or 12.962 grammes in the twenty-four hours.

In most cases the percentage of albumen does not at any time in the course of the affection exceed 0.5 per cent., and in many it does not exceed 0.2 per cent. Does the urine contain albumen in every stage of the acute inflammatory process? This question forced itself on my attention while I was engaged in watching some convalescents from scarlet fever. During the epidemic of 1853–54 I met with a few cases in which dropsy set in after scarlet fever, although the urine passed by the patients contained no albumen. In all these cases, however, very little urine was excreted—in one case, for instance, as my notes show, only two tablespoonfuls being passed in the twenty-four hours. Very soon afterwards a more abundant excretion of bloody urine set in, while the dropsy increased, and subsequently the cases followed the ordinary course of scarlatinal nephritis.

From these observations I arrived at the conclusion that the kidneys of the dropsical little convalescents from scarlet fever must have been in a condition of inflammation at the time when the urine became scanty, although it was free from albumen. I never had an opportunity of demonstrating the correctness of this conclusion by an anatomical investigation.

Quite recently, however, Henoch[1] has furnished anatomical proof of its correctness by the publication of the following case of scarlatinal nephritis:

A robust boy, twelve years of age, was admitted into the children's department of the Charité, in Berlin, on the 22d of July, 1873, with œdema of the face and scrotum, which had existed for a few days. The previous history of the boy showed that about three weeks previously he had passed through an attack of scarlet fever, and he presented on his admission distinct traces of desquamation. His urine was scanty, very acid, and deposited a sediment, but was free from albumen. The microscopical examination revealed no elements which would indicate the existence of a nephritis; the sediment consisted entirely of amorphous urates, which dissolved when heat was applied. During the two following days the œdema increased, but the urine remained unaltered. During the night of the 24th of July he had a sudden and violent eclamptic attack, with complete loss of consciousness; towards morning restlessness and attempts to get away, constant ejaculation of the words "mother," and "yes, yes," along with persistent unconsciousness and marked dilatation of the pupils. At the same time his pulse was quick, and could scarcely be felt; he was cyanotic, and his cheeks and extremities were cold. The urine, which was with difficulty drawn off with a catheter (on the 25th), contained a large amount of albumen and numerous hyaline casts, beset with fat granules.

On the 26th consciousness returned, but the cyanosis persisted, and the dyspnœa was extreme. Respiration 52. Morning temperature, 36.5° C. (97.5° F.); evening, 37.9° C. (100.2° F.). Pulse scarcely perceptible. Dullness on percussion over the lower part of the thorax, posteriorly on both sides, and very harsh respiratory sounds, with fine crepitation. Death took place on the following morning.

---

[1] Berliner klinische Wochenschrift. 1873. No. 50.

Autopsy.—Œdema of the lungs and of the aryteno-epiglottidean ligaments. Broncho-pneumonic solidification of the left lower and right upper lobes; serous effusions into the pleuræ, the pericardium, and the peritoneal sac. Extremely well-marked parenchymatous nephritis on both sides, etc.

To this case Henoch appends the description of a second case of anasarca after scarlet fever, in which the urine for two weeks sometimes contained albumen, and sometimes did not. He also reports a case observed by himself, in which no albumen was found in the urine, although the autopsy showed marked hemorrhagic nephritis. As, however, in this case the urine was not examined daily, Henoch cannot positively state that it never contained albumen and blood.

According to a quotation given by Henoch, Ferini, in an epidemic of scarlet fever which he observed, appears to have frequently found no albumen in the urine in the cases of consecutive nephritis.

To Henoch the intermittency of the albuminuria, or its complete absence in these cases, appears altogether inexplicable. If, however, we bear in mind the fact that the anatomical changes in the kidneys in this acute parenchymatous nephritis are often at the very commencement so remarkably diffuse, it seems to me that there can be no valid objection to the theory that the functions of those portions of the organ which are affected are entirely suspended, while the portions not yet involved in the process continue to secrete urine which is free from albumen. Sometimes, when the process is very widespread, there is complete suspension of the functional activity of the whole of both organs. But I am quite in accord with Henoch in regarding as suspicious every case of dropsy after scarlet fever which cannot be ascribed directly to the severe febrile affection or to the anæmia resulting from it, even when no albumen appears in the urine; that is to say, we regard it as the result of a nephritis until the contrary is established by an autopsy. I have myself never seen dropsy after scarlatina that was not due to nephritis.

Next to the constant albuminuria, an admixture with *blood* is one of the most frequent of the changes which are observed in the urine in the course of acute nephritis; indeed, a bloody coloration of the urine is sometimes the first sign of the renal affection. I have often noticed that the urine passed on one day has been free from albumen, while that passed on the succeeding day would be deeply stained with blood. The quantity of blood, like that of the albumen, varies in the different cases, and also fluctuates greatly during the course of the same case. The color of the urine at different times may vary from the lightest pale red (like meat-washings) to a blackish red. The blood may also alternately appear and disappear. One of my patients, who was attacked with nephritis after an extensive phlegmon, for weeks passed bloody urine by day and clear yellow urine by night. In the larger number of cases the hæmaturia disappears much earlier than the albuminuria. For the rest, we must not suppose that urine which is deeply tinged with blood, on that account necessarily contains a large percentage of albumen. I have tested bloody urine that was nearly blackish red in color, and found it to contain only 0.25 per cent. of albumen.

The albuminous urine of acute nephritis always contains *casts*, but these too, in different cases and at different periods of the same case, vary greatly in number. Sometimes, even in urine which contains a large amount of blood, they are so few in number that it is only after a prolonged search that a few specimens can be discovered, and again they are so numerous that several are found almost in every drop of the sediment that is placed under the microscope. In quite recent cases nearly every cast is perfectly hyaline and small, and epithelial cells from the tubuli uriniferi are often attached to them. When the urine is bloody, blood-casts are found alongside the hyaline casts. When the process is of longer standing, in addition to the recent hyaline casts we also find others which are covered with small drops of fat, and finally broad hyaline and very dark granular casts.

Besides these various forms of casts, the microscopical examination of the sediment reveals constantly more or less numerous white blood-globules, and very generally also unaltered red corpuscles or fragments of broken-down corpuscles. The red globules and their *débris*, as has been already stated, are often found in enormous quantities at the bottom of the urine glass. Sometimes we also find in the sediment considerable numbers of well-preserved epithelial cells from the urinary tubules, and granular masses, which I take to be the detritus of broken-down epithelial cells. Still I have never found either epithelium or its *débris* in such quantities as Johnson's description would lead one to expect. And now what is the physiological connection between the changes in the quantity and quality of the urinary secretion that have just been described and the anatomical alterations in the secreting organs themselves, caused by the inflammatory process? The investigations to which this question leads must, in the first place, be directed towards the vessels, as the constituent parts of the kidneys which are the first to suffer. The physiological properties of their walls are modified by the irritant that excites the inflammation, as Cohnheim has demonstrated; the vessels dilate, and from this follows the change in the circulation of the blood, and the exudation of its constituents, solid as well as fluid, which, under normal conditions, are retained in the circulating fluid by the walls of the vessels. This modification of the physiological characteristics of the vessels involves also the vascular loops of the Malpighian coils, or at least they participate in the consequent disturbance in the circulation of the blood. This is proven by the facts, which have been noticed by different pathologists, that these coils are sometimes found distended with blood in the dead body, and that blood may be effused from the vessels into the intracapsular space, whence it is carried off by the convoluted tubules (Ponfick). This, however, does not explain the invariable diminution of the urinary excretion, or the complete suppression of urine which is so frequently observed at the commencement of "parenchymatous nephritis." In the cases of which we are now speaking, the condition of affairs is not the same as in cholera, when the natural explanation of the arrest of the renal secretion is to be found in the diminution of the arterial pressure. When the inflammation is due to other causes, the blood is carried to the kidneys after its outbreak, just as it was before; it first fills the relaxed vessels to an abnormal extent, and then the secretion stops. The fact that similar functional disturbances are observed during inflammation of other secreting organs—the salivary glands, for instance—does not explain the matter. Some have endeavored to explain this diminution or arrest of secretion in nephritis upon the theory of an increase of the resistance to the secreting pressure in the renal tubules, due to swelling of the epithelium and obstruction of the tubules by fibrinous casts. In forming this theory, however, they have overlooked the fact that the diminution or arrest of secretion is observed in the very earliest stage of the renal affection, before any considerable degree of epithelial change, or any abundant deposit of fibrin in the renal tubes has taken place. Klebs ascribes the diminution or complete suppression of the urine to the already mentioned proliferation of the nuclei, between the capillary loops of the glomeruli, which he discovered in the kidneys after scarlet fever. In consequence of this proliferation the capillaries are said to be compressed, and to be rendered impervious to the circulation of the blood. Klebs himself, however, has as yet only seen this condition of the glomeruli in scarlatinal nephritis. It is very unlikely that it will ever be found to exist in every case of acute parenchymatous nephritis, in the first place, because, so far as I know, it has thus far been recognized by no other observer; and secondly, because similar disturbances of the renal functions also occur in chronic parenchymatous nephritis, in which the glomeruli certainly do not always undergo pathological changes similar to those described by Klebs.

For the present, then, we are obliged to regard the diminution in the rapidity of the circulation of the blood through the vessels, and its partial stasis in the capillaries, which are characteristic of every inflammatory process, as the special cause of the cessation of the secretion of urine in inflammation of the kidneys. We have been taught by physiological experiments on those secreting organs which admit of being controlled in the requisite manner, that the rapidity in the blood stream through these organs and the activity of the secreting process stand in direct relation to one another. Physiological research and clinical observation prove that this applies also to the kidneys. But besides the diminished rapidity with which the blood circulates through the vessels and its partial stagnation in the capillaries, we also encounter in every truly inflammatory process [1] an increased transudation of blood-plasma, and finally, an extravasation of the white blood-corpuscles from the veins and capillaries, and of red corpuscles as well from the latter. The condition of the urine shows that in parenchymatous inflammation of the kidney these processes take place not only in the nutritive vessels of the organs, which indeed no one will dispute,[1] but also in the functional vessels. The urine secreted contains albumen, white, and very generally also red blood-corpuscles—often in large quantities—the results of inflammatory transudation and extravasation through the walls of the Malpighian tuft; at the same time, fibrinous coagula or casts are formed from the albuminous urine while still within the tubuli uriniferi.

---

[1] *Cohnheim*, Neue Untersuchungen über die Entzündung, S. 63.

In my opinion it is evident *a priori* that in inflammatory processes in the kidney, not only the nutritive vessels, of which there is no question, but also the functional vessels, which are derived from the same arterial trunks, must be involved, for experience has taught us that there is a certain solidarity in the behavior of the branches in the separate vascular territories when exposed to any irritation whatsoever. Nevertheless, the view that the admixture of albumen and blood with the secretion of the inflammed kidneys must be regarded solely as the effect of an inflammatory transudation and extravasation through the walls of the secreting vessels (the Malpighian tufts) has, it appears to me, by no means met with universal acceptance. The morbid condition of the epithelium in nephritis is still constantly served up to us as the cause of the albuminuria, although no one has so far proved that the changes in the epithelial cells alone can cause the escape of any albumen at all, to say nothing of such large quantities as are often contained in the urine excreted by inflamed kidneys.

Some have appealed to the fact that no alteration has been discovered in the histological conformation of the Malpighian tufts in the inflamed kidneys. Cohnheim, in his researches into the condition of the vessels involved in the inflammatory process, was unable, even with high magnifying powers, to discover the slightest departure from their normal structure. Actual observations, however, have recently demonstrated that the blood found in the urine excreted by acutely inflamed kidneys is furnished by extravasation from the Malpighian coils. Ponfick, as has already been stated, found effusions of blood, often in considerable quantities, in the space between Bowman's capsules and the capillary tufts of the glomeruli.

---

[1] *Kelsch* is the only person who refuses to admit (loc. cit.) the existence of an inflammatory condition of the vessels in the affection which I term chronic parenchymatous nephritis.

When in favorable cases the inflammatory process in the kidneys subsides, the blood again flows through the vessels in a rapid stream, and then the previously scanty secretion becomes more abundant and as a rule excessive, so that frequently the daily quantity of urine is above the normal. The essential cause of this hypersecretion is doubtless to be found in the thinning of the blood consequent upon the retention of water during the preceding interruption of the excretion. It is well known that the rapidity of the filtration of an aqueous solution increases in proportion to the quantity of water contained in it. In consequence of this condition of the blood serum from which it is separated, and of the abnormal rapidity with which it is excreted, the now copious urine is of low specific gravity, watery, and poor in solid constituents.

The natural physiological properties of the walls of the blood-vessels are not, however, restored at once, as soon as the retrogression of the inflammatory process begins. They still remain, for a shorter or longer period, permeable for albumen, and often also for the formed elements of the blood. Months may elapse before their normal condition is re-established.

The dangers by which persons affected with acute parenchymatous nephritis are threatened consist in the reaction excited in the system at large by the disturbances of the renal functions. They are greatest at the commencement of the malady, when the urinary secretion is either completely suppressed, or is reduced to a very small quantity. They consist, in the first place, in the retention in the blood of the special constituents of the urine, and the fatal termination is ushered in by the symptoms of acute uræmia. I have seen uræmic convulsions, followed by death, after complete anuria had existed for an uncertain length of time, both in scarlatinal and in diphtheritic nephritis. I have never met with uræmic manifestations in the acute nephritis due to other causes, except when it was consecutive to cholera or pregnancy. The tendency to the development of suppurative inflammations, which has been observed in persons suffering from acute parenchymatous nephritis, must also be classed with the consequences of the retention of the urinary constituents and the contamination of the blood thus entailed. Of the fatal cases of acute nephritis which have occurred in my own practice, the ultimate cause of death, in the greater number, was purulent effusions into the serous cavities of the body, the pleuræ and the pericardium, etc.

The injurious effects of the insufficiency of the renal secretion are, however, much more frequently of a different character from those due to the retention of the nitrogenized products of the tissue waste in the blood. *The insufficient excretion of water by the kidneys leads to dropsy*—a symptom which is scarcely ever absent in the severer cases of acute parenchymatous nephritis, although very often absent in the milder cases. I have already stated that the œdematous swelling of the subcutaneous connective tissue, the hydrops anasarca, not unfrequently commences before the albuminuria sets in, in consequence of the abnormal diminution of the excretion of water by the kidneys, which is apparent even before albumen appears in the urine.

When œdema shows itself during convalescence from scarlet fever, while at the same time the urine is free from albumen, physicians have been inclined to regard it as the result of a paralysis of the cutaneous vessels, which is a consequence of the preceding hyperæmia. This explanation, however, appears to me, for many reasons, altogether unsatisfactory. In the first place, the œdematous parts do not exhibit the slightest trace of that redness which must necessarily accompany paralysis of the cutaneous vessels, in consequence of the accumulation of blood in them, due to the diminished resistance presented by their walls; on the contrary, the skin of the swollen parts is invariably remarkably pale, often even before the swelling begins. In the second place, in every case of dropsy after scarlet fever which I have observed, the œdema was preceded by complete suppression, or very abnormal diminution of, the urinary secretion. In those cases in which no albumen was contained in the urine at the time the anasarca first began, it invariably made its appearance after a very short interval, as in the case of nephritis following acute articular rheumatism, reported above (Case XII.). Thirdly, if the œdema is the result solely of the cutaneous hyperæmia, it ought to occur with equal frequency after other affections of the skin, in which the congestion is as intense as in the scarlatinal exanthem, viz., erysipelas, confluent small-pox, etc. Experience, however, has taught us that this is by no means the case. Fourthly and lastly, a certain correspondence would then exist between the intensity of the scarlatinal rash and the probability of a dropsy setting in, which no careful observer would be willing to admit.

In every case of acute parenchymatous nephritis, be its origin what it may (always excepting, however, the nephritis of cholera), the dropsy is dependent upon the interruption of the urinary secretion. This fact can be demonstrated, without exception, in all cases which can be watched from the commencement. The more complete the interruption of the secretion, the more rapidly is the dropsy developed; when the anuria is complete, it may be developed within a very few days.

When a very abundant excretion of urine is again re-established, the œdema disappears with corresponding rapidity; it may thus be of very short duration when a severe renal disorder subsides rapidly, and may last for many weeks under other circumstances.

The dropsy in acute parenchymatous nephritis differs in no respect from that which occurs in other renal affections. It begins almost without exception in the loose subcutaneous cellular tissue, and affects that tissue principally.

As a great many patients are confined to their beds at the time the dropsy commences, the œdema frequently appears first of all in the back, in the lumbar regions; in other cases it appears first in the face. Not unfrequently a puffiness of the eyelids furnishes the first intimation of the existence of the renal disease. Enormous accumulations of water also occur in the serous cavities of the trunk. Œdema of the lungs or of the glottis is more uncommon.

In the milder cases of acute parenchymatous nephritis there is oftentimes not a trace of any œdematous swelling; I have frequently found it absent even in cases in which the urine was tinged with blood for a considerable time. The fact that dropsy is not observed with equal frequency in the forms of acute nephritis which differ only in their etiology, probably depends upon the intensity of the action which the exciting agent of the inflammation exerts on the vessels of the kidneys. For instance, it is much less common in the nephritis of diphtheria than in that of scarlet fever. I have only encountered it once in connection with diphtheria in a case in which during the last five days of life not a single drop of urine was passed. Herr Kardel, who called me to see this patient, assured me, however, that in his country practice he had met with many similar cases. In nephritis following exposure to cold, dropsy almost invariably occurs.

The dropsy in acute nephritis becomes dangerous to life principally in consequence of the mechanical disturbances of the respiration which it may occasion; it may cause excessive accumulations of fluid in the thoracic cavities, or œdematous infiltration of extensive portions of the pulmonary parenchyma; or, finally, in rare cases œdema of the glottis.

Gangrenous destruction of extensive portions of the skin, such as often occurs in chronic renal affections, I have never met with in acute parenchymatous nephritis. The reaction upon the nutrition of the general system must also be mentioned as an indirect result of the acute parenchymatous nephritis. Unquestionably the general malnutrition depends partly upon the disorders of digestion which accompany many of the graver cases of this disease.

At the commencement we occasionally observe severe and obstinate vomiting, a reflex symptom provoked probably by the irritation of the nerves in consequence of the acute swelling of the kidneys. When vomiting sets in at a later period of the disease, it may be uræmic in character.

Loss of appetite invariably accompanies inflammation of the kidneys when it is attended by any febrile movement, and is occasionally an obstinate symptom even when there is no fever; diarrhœa occurs much less frequently.

The influence of the primary disease which has occasioned the acute renal inflammation, and also the direct influence of the derangement in the secretion of the urine, are of far greater importance for the production of the general disturbance of nutrition and loss of strength, than these incidental derangements of the digestive organs. It is sufficiently well known that scarlet fever, diphtheria, etc., etc., reduce the general system in proportion to the severity of the fever by which they are accompanied; and that individuals who have passed through these maladies are often left in an exceedingly anæmic condition. This effect of the diseases we have mentioned is increased when they are followed by a nephritis, in consequence of which considerable quantities of albumen and of blood are discharged through the kidneys for weeks or even months. The patients then become still paler and weaker, and often very rapidly become emaciated. In many cases, it is true, this emaciation is concealed by the general anasarca.

Nevertheless, even those cases of acute parenchymatous nephritis which are not dependent upon an antecedent febrile affection—for instance, those which are due to catching cold, exert an injurious influence on the strength and general nutrition of the system, although the febrile symptoms by which the malady is ushered in are always of short duration, and can scarcely be of any consequence in this connection.

The continuous loss of albumen and blood in the urine is undoubtedly the principal factor in the production of the rapidly developing anæmia, loss of strength and emaciation, which are observed even in the uncomplicated cases of acute parenchymatous nephritis. In this connection, I must insist particularly on the fact that in every severe case not only the albumen of the serum, but also large quantities of the formed elements of the blood—the corpuscles—are lost.

Prognosis.

The prognosis of acute parenchymatous nephritis depends pre-eminently upon the nature of the exciting cause; in the first place, because some of the exciting causes are liable to be followed by other grave complications besides the renal affection, or, even before the commencement of the latter, may themselves occasion great disturbances of the general nutrition and much prostration of strength, while in other cases the agent that excites the inflammation produces no changes in any other organs than the kidneys.

Among the causes which belong to the first category we may mention scarlet fever, and among those of the second, catching cold. There is also another way in which the nature of the exciting cause exerts an influence on the prognosis, for experience has shown that the cases of renal disease which are due to certain determined causes, run a more severe course than those which owe their origin to other causes.

I have already mentioned this fact, and expressed the opinion that one cause may occasion a more profound impairment of the physiological integrity of the renal vessels than another. This hypothesis of course does not exclude the possibility of differences in the intensity of the action of the same cause; we sometimes meet with mild cases of nephritis after exposure to cold, and with very severe cases after diphtheria. This, however, does not invalidate the general rule.

Of all the etiologically distinct forms of nephritis which we have been discussing, the most dangerous is unquestionably that which succeeds scarlet fever. This, however, is entirely due to the fact that it so frequently attacks individuals who are already greatly debilitated; or else is complicated with grave affections of other organs, which are likewise dependent upon the scarlatinal process: as for example, sloughing of the cellular tissue in the neck; phagedenic ulceration of the tonsils or soft palate; diphtheritic inflammation of the nares, or of the middle ear, etc. When scarlatinal nephritis attacks an individual who is still vigorous, and runs its course unattended by any grave complication, it is of itself far less dangerous. But apart from the individual condition of the patient, the character of the epidemic and the nature of the patient's surroundings exert an influence upon the relative mortality of scarlatinal nephritis.

Out of twenty-two cases treated by me in the year 1853–54, partly in my polyclinic and partly in my private practice, I lost only five; while every one of the thirteen cases treated in my polyclinic in the year 1863 terminated fatally. Between and since these years, I have treated a considerable number of cases both in the hospital and in private practice, but have not lost a single one of them. I have never met with a fatal case of inflammation of the kidneys after measles, rubeola, or small-pox. Indeed, I have only once met with this affection as a sequel of small-pox; elsewhere also it appears only to have been observed in connection with the hemorrhagic form, so that the fatal termination should be ascribed not to the renal affection, but to the malignancy of the variolous process. All the cases which followed measles and rubeola in my practice ran a mild course and speedily recovered.

The nephritis of diphtheria, when the patient does not succumb to the original malady, as a rule runs an equally mild and speedy course. Out of the large number of cases of diphtheria which I have unfortunately had the opportunity to observe here in Kiel, only one died from the subsequent nephritis, although in hardly any of the severer cases did the kidneys remain entirely unaffected. The experience of my colleague Kardel, who has been frequently mentioned in this article, has, however, been less favorable.

The nephritis which is consecutive to acute inflammation of the skin and the subcutaneous areolar tissue, pursues a favorable course, and recovery rapidly ensues when the primary affection—as, for example, erysipelas—is of short duration. When, however, extensive phlegmons or other suppurative processes in the areolar tissue of long duration are present, the symptoms of the renal affection produced by them also persist for a long time. In one of my cases of peripleuritic abscess the secondary nephritis was in fact the immediate cause of death.

All the cases of acute nephritis dependent upon extensive burns, that I have met with, terminated fatally, but death ensued so rapidly that I could not regard it as actually due to the renal affection alone. In five cases I have seen nephritis occur as a complication of endocarditis. In three of these cases the endocarditis accompanied acute articular rheumatism; in the other two cases, the endocarditis was ulcerative and was not complicated by any joint-affection, and in both the nephritis was the cause of death. In two of the first three cases the renal affection became chronic; the third case was cured. This patient, however, was attacked a few weeks later with facial erysipelas, and the renal inflammation returned, but quickly disappeared again.

With regard to the influence of the inflammation of the kidneys upon the mortality of relapsing fever, I can say nothing from personal knowledge. Of the two cases which I have observed in the course of abdominal typhus, one died of pneumonia, and the other recovered rapidly. The only case I ever saw occur as a complication of croupous pneumonia recovered after lasting for two months.

None of my cases of nephritis which were clearly due to exposure to severe cold died during the acute stage. In all of them, however, the symptoms were severe, and the disease followed a protracted course; one, after beginning acutely, became chronic and led to extreme dropsy, which lasted for a long time. The patient was subsequently able to pursue his avocation for two years, although suffering from persistent albuminuria, and finally died of purulent pericarditis.

However, in all cases of acute parenchymatous nephritis, whatever the cause may be, the rule holds good that either recovery or death sets in within a relatively short period. Taking all the cases together, recovery is decidedly by far the more frequent issue.

The transition to a chronic disease, a chronic renal affection, is exceptional; it seems to occur most frequently in the cases that follow exposure to cold, and perhaps also in the cases of true rheumatic nephritis. The reason why the exception occurs in these instances is by no means clear. In my cases of rheumatic nephritis the disturbances of the circulation caused by the valvular lesions of the heart, which were developed at the same time, ought perhaps to be taken into account.

Apart from the individual conditions and complications, the prognosis may be set down as absolutely bad, so far as my own experience goes, in every case (with the exception of cholera nephritis), in which the suppression of urine is complete. In no such case have I ever seen recovery take place; but then I have only seen complete suppression occur in scarlatinal and diphtheritic nephritis. A case of nephritis after scarlet fever, observed by Biermer, and published in the nineteenth volume of Virchow's Archives, teaches us, however, that it is possible for the secretion of urine to be resumed, after it has been repeatedly suppressed for several days at a time. The patient in question finally died with uræmic symptoms. The fatal termination in the cases of complete anuria that have come under my observation was not always ushered in by uræmic symptoms; in several cases death was not preceded by any violent symptoms, the little patients dying quietly after having previously become excessively dropsical.

I never witnessed complete anuria in an adult as the result of acute nephritis (always excepting cholera nephritis). The most severe uræmic attacks do not invariably prove fatal, even in children, as the following case proves.

Case XIII.—On the 16th of July, 1866, I was sent for to visit a little girl, nine years old, whose brothers and sisters had suffered from scarlet fever a few weeks previously, while my little patient herself had at least apparently escaped the contagion. Eight days before I was called in, the parents had perceived a sudden swelling of her face and extremities, and especially of the stomach. For two days the child had kept her bed. When I saw her, she was pale, her entire body was swollen, and she had a great deal of ascites. Pulse slow and remarkably tense. Temperature normal. The urine, which was very scanty, was deeply tinged with blood, contained a good deal of albumen, and in its sediment, besides an abundance of red blood-corpuscles, a good many blood- and hyaline-casts, to most of which renal epithelial cells were still attached, were found. Hot baths were ordered to be repeated every day, along with a potash draught (Kalisaturation). The dropsy increased. I could obtain no exact information as to the quantity of urine excreted daily; I was obliged to content myself with the knowledge that but little urine was passed. I was only able to see the child at long intervals, as the child did not live here.

On July 22d the child suddenly lost her sight, and shortly afterwards general convulsions came on, which were followed by insensibility. During and after the attack enormous quantities of frothy mucus are said to have been discharged from the mouth; and even after consciousness was restored, large quantities of a watery fluid were vomited up. The complete blindness continued. The anasarca continued to increase steadily, and the general convulsions recurred again on the 24th of July, and were followed by coma, which lasted for twelve hours. The consciousness then returned, and a few hours later vision was also restored quite suddenly. At the same time a copious perspiration, and soon afterwards an abundant excretion of urine, set in. Within a few weeks every trace of dropsy had disappeared. On the 5th of September I saw the patient for the last time; she was perfectly well, and her urine did not contain a trace of albumen. I have been unable to obtain a more minute account of the history of this case, and was unable to especially make any accurate examinations of the urine, because I was only able to see the patient at long intervals.

The degree of the functional inactivity of the kidneys furnishes the true measure of the extent of the impending danger in each individual case. So long as the excretion of urine remains quantitatively far below the normal, there is great danger of the occurrence of uræmic attacks; and the development of dropsy, or its increase if it has already set in, is unavoidable. Although the latter is not so very frequently the sole, immediate cause of death, still an extensive œdema of the lungs is not exactly a rare cause of death in acute nephritis; the fatal result, however, is much less frequently due to œdema of the glottis or to excessive dropsical effusion in the serous cavities.

When, in the course of an acute nephritis, purulent effusions take place into the serous sacs or into the meninges, or an inflammatory infiltration of the pulmonary tissue sets in—lesions which are often accompanied by active febrile movement—the case, according to my own previous experience, is as good as certain to terminate fatally.

Diagnosis.

The diagnosis of the acute parenchymatous inflammation of the kidney must depend in the first place upon the etiology. When, after one of the above-mentioned diseases, albuminuria and hematuria set in, and are followed by dropsy, the diagnosis of an acute nephritis is positively established.

We must not allow ourselves, however, to regard every albuminuria that presents itself during the course of a severe febrile affection as an indisputable sign of a commencing inflammation of the kidneys, even though some hyaline casts be discharged with the albuminous urine; even in a scarlet fever patient this does not always indicate the beginning of a nephritis, while it occurs almost without exception in every case of diphtheria that is at all severe, is not often absent in severe cases of typhus, and is common in small-pox. Although, as has already been stated, nephritis is a rare sequel of the last-mentioned processes, we are only justified in ascribing the changes in the urinary excretion to an inflammation of the kidneys, when at the same time the excretion of urine is notably diminished in quantity, or when in addition to the hyaline casts an abundance of blood-casts and free red blood-corpuscles are found in the sediment deposited by the cloudy and scanty urine. In such a case we will not, as a rule, have long to wait before dropsy sets in, if it has not already appeared.

But even when bloody urine is passed by a feverish patient, it does not necessarily follow that nephritis exists. In nearly every case of hæmaturia which I have had an opportunity to observe in variolous patients, the source of the hemorrhage was not the renal substance, but the mucous membrane lining the pelvis of one of the kidneys. Dr. Carl Oscar Unruh,[1] of Dresden, met with 28 examples of extensive hemorrhage from the pelvis of the kidney out of 212 autopsies upon cases of small-pox. I was able to refer to the same source the hæmaturia in several cases of Werlhoff's purpura disease, which ran a rapidly fatal course, with excessive elevation of temperature.

The characteristic signs by means of which the true renal hæmaturia may be differentiated from the bloody urine which is due to affections of the mucous membrane of the urinary passages, have been already described in the chapter on Hæmaturia.

We may find it difficult to decide whether an attack of acute hemorrhagic inflammation has involved previously sound kidneys, or organs which were already the seat of a chronic diffuse affection. I have never known this complication to occur in kidneys which were in a state of advanced amyloid degeneration, but have met with it in chronic parenchymatous nephritis and in the cirrhotic kidney. Chronic parenchymatous nephritis may be developed from an acute attack; when it begins, as it usually does, insidiously, it invariably produces extreme anæmia and dropsy. The dropsy may give way and the patient pick up wonderfully, although the albuminuria still persists. In such cases it not unfrequently happens that an aggravation of all the symptoms is ushered in by a fresh attack of acute hemorrhagic nephritis. The history of the case alone can then enable us to interpret the process correctly, for the results of the examination of the urine under these circumstances do not suffice to explain the condition of affairs.

---

[1] Ueber Blutungen in Nierenbecken und Ureteren bei Pocken. Archiv für Heilkunde. 13th year. 1872. p. 289.

An intercurrent attack of acute nephritis is much less frequently observed in the course of cirrhotic atrophy of the kidney. In a case of this nature which came under my observation the quantity of urine excreted, which had previously been very abundant, suddenly diminished under febrile symptoms, and fell far below the normal average; the urine was bloody, but had a low specific gravity. The case proved fatal by uræmic convulsions. Even without any knowledge of the history, the existence of a renal atrophy in this case, before the hæmaturia began, might perhaps have been surmised from the hypertrophy of the left ventricle and the extreme tension of the arterial pulse. To the cardiac hypertrophy an endocarditis involving the mitral valve was added, which was developed simultaneously with the nephritis. I believe, however, that in other cases where the patients, despite the existence of renal atrophy, had been in the enjoyment of apparently perfect health previous to the development of the complicating nephritis, the diagnosis may be surrounded by great difficulties. The main evidence for the demonstration of a chronic renal affection must always be sought for in the state of the circulatory organs. An unyielding pulse may be present even in simple acute nephritis, but a perceptible enlargement of the left ventricle is evidence that some more protracted influence has been at work.

Treatment.

In very many cases it is not the renal inflammation alone that we have to consider in our treatment, but also the complications; in many instances a proper attention to the latter fulfils in a certain sense a prophylactic indication with respect to the renal affection. This is the case, for example, when proper attention is paid to securing an early and sufficiently free outlet for the pus and to keeping the cavity of the abscess clean, in cases of extensive and acute suppuration of the areolar tissue. At the same time I do not wish to maintain the applicability to all cases of Fischer's hypothesis as to the influence of the acid fermentation of pus and its products upon the development of renal inflammation. On the contrary, my experience has shown that inflammation of the kidneys may be associated as a secondary affection with very acute suppurative processes in the areolar tissue, before the pus, which at a later period was evacuated by an incision as an odorless, creamy fluid (a pus bonum et laudabile), was exposed to the action of the external air, and consequently before it was placed under the conditions necessary for the development of the acid fermentation. Further, my experience is that the renal affection in these cases rapidly gets better, as soon as the pus is evacuated as completely as possible, and its re-collection, and consequently also its decomposition or fermentation, is prevented. *In a case of acute nephritis it is necessary to keep the patient in bed if we wish to render the prospect of a rapid and favorable course more certain.* But while premising this statement I must expressly protest against being supposed to uphold the view that every case of acute nephritis is to be ascribed to catching cold. By maintaining a uniform and constant warmth of the skin, however, we avoid those fluxions to the kidneys which, as physiological experiments show, are associated with the cooling of the cutaneous surface. Healthy kidneys suffer no injury from such fluxions, for they possess, in the power of pouring out an increased secretion, the means of at once equalizing the increase of the blood-pressure. In the inflamed kidney, however, this correcting power is wanting in a degree that depends upon the intensity of the functional disturbance, and hence the result of the fluxion must be an increase of the inflammatory hyperæmia and of the exudation.

In the great majority of cases, those who are attacked with acute nephritis are already reduced in general condition by an antecedent severe febrile affection or by some drain upon the blood, and to this is now added the continuous loss in the urine of more or less excessive quantities of albumen, and usually also of blood. For this reason the patients, as a rule, in a very short time become anæmic, and rapidly lose strength and flesh. The object of the treatment, therefore, is to maintain the strength and improve the general nutrition. Unfortunately, the condition of the digestive organs renders the fulfilment of this indication in many cases difficult; it is often difficult to supply the patient with aliment which can be sufficiently assimilated by the digestive organs. A milk diet, with all its possible variations, would be the ideal bill of fare for a patient of this kind. Here, in North Germany, where milk is still easily obtained even in the towns, the customs of the country help us, for milk and buttermilk, soups with rice, barley, and oatmeal grits, are among the favorite and ordinary dishes of the people. When the state of the digestive organs and the patient's appetite will permit, a more solid diet, consisting of easily digestible meats, light vegetable food, and good bread, may, of course, be adopted. As drinks besides water, milk, buttermilk and lemonade may be taken, and, when the patients are very much enfeebled, a moderate quantity of good red wine may be recommended. On the other hand, I forbid the use of tea or coffee—substances which are supposed, and certainly not without reason, to exert an irritating action on the kidneys.

The same reasons which, from the very commencement of an acute nephritis, render it necessary for us to provide for the proper nourishment of the patient, forbid just as decidedly the employment of any measures which would curtail the amount of nutritive material in the body or hinder its renewal. I must in the most positive terms deprecate the employment, simply as a means of fulfilling the indicatio morbi, of bloodletting in any form, or of the treatment by purgatives, which is still in vogue in some places. In my younger years I employed these different modes of cure boldly enough myself, and know right well that a patient convalescing from scarlet fever will not necessarily die from the application of a few leeches or wet cups ; and also that a robust young blacksmith, who has been attacked with acute nephritis in consequence of exposure to cold, will bear a venesection well. My own experience, however, has taught me that bloodletting and purgation, even when continued until a noticeable effect is produced, will neither cure the inflammation of the kidneys nor shorten its course, while, on the other hand, they greatly depress the patient's strength. We will presently have occasion to speak of the usefulness of bloodletting in the treatment of the indicatio symptomatica.

The so-called remedia antiphlogistica interna do not exert the slightest influence upon the inflammatory process in the kidneys. From tartar emetic I have seen no other result than an increased disturbance of the digestive functions, and calomel has proved equally useless. I have never tried the other mercurial preparations.

It is still customary in diffuse inflammation of the kidneys to administer such drugs as one may expect to be reabsorbed, more or less unaltered, from the digestive tract, and to be excreted again by the kidneys, and to which astringent properties have been ascribed. It is assumed that such medicines, in their transit through the renal vessels, will cause the walls of these vessels to contract. They consist principally of gallic acid and drugs which contain tannin. I have never seen any satisfactory results follow the use either of pure tannic acid or of the decoction of uva ursi, and hence have long since ceased to employ them in acute parenchymatous nephritis.

The only means known to me by which, without depressing the general nutrition, the congestion of the kidneys can be diminished, and thus a direct impression be made upon the inflammatory process, consists in the constant maintenance of a state of cutaneous hyperæmia. It was with a view to this that I insisted on strict rest in bed, while speaking of the dietetic rules which should be enforced in the treatment of nephritis. Among the curative measures, a sustained diaphoresis deserves unquestionably the first place, not only in the symptomatic treatment of the dropsy, but as a measure which favors and promotes the retrogression of the inflammatory process in the kidneys.

The employment of diaphoresis in the treatment of renal inflammations has been, as is well known, in general use for a long time, but it has been employed mainly as a symptomatic measure against the annoying dropsy. At all times, however, while ordering hot baths in affections of the kidney, physicians seem to have had in mind the antagonism between the skin and the kidneys, and the possibility of relieving the renal vessels by means of an artificially produced hyperæmia of the skin, and of combating in this way the inflammatory process in the kidneys. I have already referred to the experiment by which Koloman Mueller recently demonstrated the correctness of this opinion.

We possess, as is well known, an exact measure of the degree of the blood-tension in the renal vessels, in the rapidity with which the kidneys secrete urine. Mueller established fistulous openings into the ureters of dogs, then shaved off the hair, and by counting the number of drops flowing from the canules which had been introduced into the ureters, ascertained the quantity of urine secreted each minute. Taking this quantity as the normal, he found that more than the normal quantity was secreted, that consequently the blood-pressure in the renal vessels was increased, whenever, by means of affusion with cold water, the shorn skin of the animal under observation was cooled to any considerable extent; that, on the contrary, an abnormally small quantity of urine was excreted—that consequently the blood-pressure was diminished in the renal vessels—whenever the skin was warmed by affusion with hot water or by the application of hot cloths. Standing in apparent contradiction to the results of Mueller's experiments is the fact ascertained by me by careful observation of a great number of cases, that the production of active diaphoresis (by means of hot baths) in an acute inflammation of the kidneys, which had caused great diminution of the urinary secretion, is followed at once by a marked increase instead of by a still further reduction of the renal secretion. Koloman Mueller, however, experimented on animals with healthy kidneys, through the vessels of which the blood circulated in a normal manner. My observations, on the contrary, were made on patients through whose vessels the circulation of the blood was obstructed by inflammatory stasis, in consequence of which the process of secretion was arrested. In my patients, the artificially produced congestion of the cutaneous vessels necessarily helped to bring the stagnant blood in the renal vessels again into circulation, since the natural obstacle presented to their disgorgement by the distention of the general venous system must necessarily have been diminished by the diversion of large quantities of blood to the surface of the body. It is in this way, as I believe, that the induction of a diaphoresis contributes to the re-establishment of the normal condition of the circulation through the inflamed kidneys, and thereby also to the re-establishment of the normal secretion. In this sense an efficient diaphoretic treatment fulfils not only the indicatio symptomatica, but actually also the indicatio morbi in a better and surer manner than does any other method of treatment. With regard to the methods that may be employed for this end, I have already expressed my views above (at page 219 et seq.), when discussing the treatment of dropsy from obstructive hyperæmia of the kidney.

Of the symptoms which require special attention in the treatment, the first in point of importance is the *complex of uræmic manifestations*. The attention of the physician when treating a case of acute nephritis must always be directed to this danger. The outbreak of a uræmic attack is sometimes preceded by complete anuria, but more usually only by a considerable diminution in the excretion of urine and of urea. The observant and careful physician will therefore find himself forewarned in time to work against the impending danger, and will endeavor to eliminate by some other channel the constituents of the urine that are retained in the body in consequence of the arrest of the renal functions. For this purpose, too, I regard prolonged hot baths with subsequent packing, the object of which is to excite profuse sweating, as the treatment which merits the greatest confidence, not only because, as I have just endeavored to prove, we have reason to expect therefrom the re-establishment of the normal conditions of the circulation and of the secretion in the kidneys, but also because in the perspiration urea as well as water is removed from the body. This takes place even under physiological conditions, according to the testimony of reliable authorities,[1] and under pathological conditions has been observed in an especially marked degree by others as well as by myself.[2] I must, it is true, admit that the employment of hot baths occasionally hastens the outbreak of uræmic convulsions. An example of this is presented by Case VII. The same case, however, shows that the treatment nevertheless ultimately exerted a beneficial influence on the general condition of the patient, since it was speedily followed by an abundant secretion of urine. It is only in those cases where a high febrile temperature coincides with the arrest of the renal secretion that I would hesitate to put the patient into a hot bath, and would confine the diaphoretic treatment to packing in wet sheets. In view, however, of the dangers which overhang the patient when the suppression of urine is complete, the employment of other means of producing derivation should not be neglected. We should not hesitate to employ the *drastic purgatives.* Both experiment[3] and clinical experience teach us that when the renal activity is diminished, urea as well as water can be excreted by the intestinal mucous membrane, and that this substance, as a rule, undergoes ammoniacal decomposition as soon as it enters the digestive tract. Among the purgatives which rapidly produce profuse watery stools, I may recommend the preparations of senna, colocynth, gamboge, etc. Watery discharges are, however, readily produced in acute nephritis. The least reliance should be placed upon the action of the diuretics proper, when the object we have in view is to avert the danger incident to the arrest of the renal secretion in a case of acute nephritis.

The drastic diuretics, and those which contain volatile oils, should be avoided on account of their irritating action on the kidneys, from which we apprehend, either with or without reason, an increase of the inflammation. As far as I know, however, no one has reported any positive experiences bearing on this question. The saline diuretics, such as the various potash salts, in combination with vegetable acids, have proved to be of but little if any efficacy in counteracting the severer functional disturbances in acute nephritis. On the other hand, I have frequently observed a decided and remarkable increase of the renal secretion in patients who had acute nephritis, with high fever, to whom I gave a solution of potash in an infusion of digitalis.

---

[1] Dr. *W. Kuehne*, Lehrbuch der Physiol. Chemie. Leipzig. 1868. S. 433. *Leube*, l. c.

[2] See p. 107.

[3] *Bernard* et *Barreswill.* Leçons sur les propriétés physiologiques et les altérations pathologiques des liquides de l'organisme, par M. C. Bernard. 1859. Tome 2me, p. 36 et seq.

When uræmic convulsions make their appearance the patient is at once placed in immediate danger of death. In consequence of the uncertainty and obscurity which, even at the present day, surround the immediate cause of these symptoms, it is impossible to lay down rational rules for their treatment. We are thrown back, therefore, upon empirical means in this point. What should be done or left undone in particular cases will, moreover, often depend upon collateral circumstances, all of which it would be impossible for me to consider here. Our medical forefathers, who in eclamptic attacks—which are often enough due to other causes than an insufficient renal excretion—saw only a nervous symptom, which they ascribed to cerebral congestion, sought to cure it by the employment of derivatives from the head, and of remedies calculated to soothe the excited nervous system. General depletion of the vascular system by venesection, local abstraction of blood from the head, blisters and mustard plaster to the nape of the neck and the extremities, fulfilled one indication, and opium the other.

The methods of treatment which have been recommended from all sides for this dangerous symptom, since the etiological connection of many cases of eclampsia with renal diseases was discovered, have been as manifold as the views of physicians with regard to the essence of uræmia. Those who believe in the ammonia theory would have us convert the poisonous alkali into a harmless salt by administering acids; for this purpose either hydrochloric or the vegetable acids which, as we know, can pass into the blood, have been recommended. Favorable results were expected particularly from the employment of benzoic acid. Others prefer even yet to make use of the intestinal canal, and recommend the administration of drastic cathartics in order to remove the poisonous urinary constituents which are retained in the blood.

With benzoic acid, from which so much was expected, I have never obtained the slightest good result, although I have been by no means timid in its employment. At the most large doses of it (0.5 gramme [seven and a half grains] repeated every three hours) have caused diarrhœa.

Purgatives I certainly consider are particularly indicated, and I employ them regularly as soon as uræmic convulsions occur in a case of acute nephritis, provided profuse spontaneous diarrhœa has not, as it occasionally does, preceded their development.

In such a critical state of affairs, however, especially when the attacks of convulsions, any one of which might prove fatal, are rapidly repeated, we should not, it appears to me, rest content while waiting for the action of the purgatives.

The first case of eclampsia which I saw as a clinical assistant occurred in the person of a primipara with renal disease, and the late Michaelis, who was then my preceptor, made me bleed her freely. The patient, who had previously recovered from one attack of convulsions only to fall into another, had only one severe convulsion after the venesection. The delivery was then completed, and the patient rapidly recovered and was quite well in a very short time. In similar cases I have abided faithfully by this line of practice, and have never had occasion to regret it. When, in cases of acute nephritis, we have to deal with individuals who are still tolerably strong, I consider that *venesection is indicated* as soon as uræmic convulsions occur; these accidents happen by far most frequently at the commencement of the malady, before the patient is much reduced.

The quantity of blood which is to be taken must be determined by the state of the nutrition and general strength of the patient; a very robust adult will bear the loss of from three hundred to five hundred c.cms. (from ten to seventeen fluidounces) without difficulty. In children we must content ourselves with the application of a number of leeches corresponding to the years of the patient, and they should be placed on the head, according to the old practice. The question whether the improvement that has, in my practice, followed bloodletting in uræmic attacks is attributable to the mere lowering of the arterial blood-pressure, or to the reabsorption of pathological effusions in the cranial cavity thereby promoted, or finally, to the removal of some portion of the pernicious substances from the blood, must for the present remain unanswered.

The application of blisters and mustard poultices I believe to be useless, and even under some circumstances harmful, since in the cases that recover they may entail troublesome cutaneous affections. On the other hand, I look upon the introduction of anæsthetics in the treatment of eclampsia as a great acquisition. They act more rapidly than opium and its preparations, and do not exert the same injurious effects on secretion that in our cases have attended the use of the latter. Instead of chloroform inhalations, the hydrate of chloral should always be employed, whenever it can be administered to the patient, either by the mouth or by a clyster. The dose must be determined by the patient's age. To children a gramme (15 gr.) may be given; to adults as much as three grammes (45 gr.). The remedy may be repeated if necessary, and may be given without fear even to anæmic individuals who would not bear bloodletting.

This is essentially the treatment followed in earlier times. So far as practice is concerned, the theoretical controversies over the nature of uræmia have proved utterly barren. The symptom which, in acute nephritis, most frequently demands therapeutic consideration is the dropsy. In this particular all that I have said about the symptomatic treatment of dropsy in obstructive hyperæmia of the kidneys holds good. There, as well as in acute nephritis, a methodical diaphoresis is the sovereign remedy for the dropsy.

When the acute nephritis is from any reason accompanied by sharp fever, antipyretics may be indicated, such as quinine and digitalis. In the treatment of the secondary inflammations of the pleura, the pericardium, etc., etc., we are restricted to the use of the same remedies, together with operative measures when they are indicated.

The reflex vomiting that is occasionally present at the commencement of the nephritis yields most rapidly to the internal administration of pieces of ice, which the patient should be allowed to swallow ad libitum. Small doses of morphia, either subcutaneously or given internally in cherry-laurel water, sometimes prove effective against this symptom. Should uræmic vomiting or diarrhœa set in at a later period, we may have recourse to warm baths again; but they are then usually entirely ineffective. When the patient's digestive organs perform their functions well and regularly, we do not need to pay any special attention to the re-establishment of the strength; but when a marked degree of anæmia is present, the use of ferruginous tonics is indicated. In view of the fact that the digestive organs in these cases are usually very irritable, it will be advisable to select the mildest preparations of iron, *e.g.*, some salt of iron with an organic acid.

Supplement.

The Acute Parenchymatous Nephritis of Pregnancy.

Although Rayer[1] had previously announced that he had repeatedly observed his néphrite albumineuse in pregnant women, and had also made mention of a case published by Martin Solon, Lever[2] was the first to trace the causal connection between the epileptiform attacks, long known to accoucheurs and which were so frequently dangerous to the puerperal patients, and the excretion of albuminous urine. Rayer does not speak decidedly concerning the connection between pregnancy and renal disease; in the short notice with which he prefaces the reports of the cases observed by him, he says that the pregnant women in whom he recognized the renal affection had either contracted a severe cold by exposure when their bodies were overheated, or had been exposed to the habitual influence of cold and moisture in unhealthy habitations. Farther on, however, he adds the remark that the coincidence or complication of pregnancy with nephritis demands careful investigation. Nevertheless, he states positively that when the renal inflammation sets in for the first time in the latter months of pregnancy, it is much less dangerous than the same affection when it exists before conception and continues throughout the pregnancy, or when it makes its appearance in the early months of this condition. Among German writers, Frerichs was the first to devote special attention to the nephritis of pregnancy. He ascribes its development to the action of two etiological influences: first, the alteration of the blood-crasis caused by pregnancy; and second, the mechanical interference with the venous circulation in the abdomen.

---

[1] *Rayer*, l. c. Bd. 2. S. 399.

[2] Guy's Hospital Reports. 1843.

The blood of the pregnant woman is usually more watery and richer in fibrin than normal blood; the quantity of albumen and of red blood-corpuscles is diminished in it, while the white corpuscles are more numerous. Frerichs places this alteration in the constitution of the blood in pregnancy by the side of that "crasis" on which depend those cachectic conditions that are produced by a long-continued discharge of albuminous materials (suppuration, ulceration, etc.), and he ascribes it to the impoverishment of the maternal blood consequent upon the drain of nutritive materials required for the nutrition and development of the fœtus. With regard to the influence of the interference with the venous circulation in the abdomen of the pregnant woman, Frerichs does not express himself clearly and decidedly. He merely says:[1] "The change in the shape and the position of the uterus is by no means always absolutely the same; it may leave the circulation through the renal veins entirely unobstructed, and fortunately this is the ordinary rule; but, on the other hand, it may under other circumstances cause albuminuria and degeneration of the kidneys in just the same way as do valvular lesions of the heart." Lever expressed himself as decidedly of the opinion that the pressure of the pregnant uterus upon the renal vessels is the cause of the affection of the kidneys. Rosenstein[1] upholds the same view, and says: "The pressure which, under conditions that are not thoroughly known to us, the enlarged uterus in the latter months of pregnancy occasionally exerts upon the vessels of the abdominal organs, and especially upon the renal veins, obstructs the circulation of the venous blood, and in this way obstructive congestion of the kidneys, as well as of other abdominal glands, is produced." He accordingly discusses the renal affection of pregnancy in the chapter on the obstructive hyperæmia of the kidneys. It must, however, at once strike the student that not a single iota of evidence founded upon fact can be adduced to establish this altogether gratuitous assumption of a compression of the renal veins by the pregnant uterus. The clinical symptoms are by no means the same as those of obstructive engorgement, and the anatomical appearances not only do not resemble those of cyanotic induration of the kidneys, but correspond in every particular with those of parenchymatous nephritis.

---

[1] L. c. p. 219.

The position of the renal veins protects them under all circumstances from any direct pressure on the part of the pregnant uterus. Any one who has had the opportunity of opening the body of a woman far advanced in pregnancy, and has observed the relative positions of the abdominal organs, could not fail to perceive how inadmissible is the theory that a mechanical pressure is exerted upon the renal veins by the distended uterus.

The uterus, the lower end of which is concealed in the pelvic cavity, would have to be bent backward on itself at a considerable angle just above the inlet to the true pelvis (a flexion which, as is well known, would be prevented by the round ligament), in order to be able to touch with its posterior wall any part of the anterior surface of the second lumbar vertebra. Even if it should reach this position it might, it is true, compress the left, but it could never affect the right renal vein.[2] In two autopsies which I held on the bodies of women far advanced in pregnancy, I found the uterus held in close contact with the anterior abdominal wall by the round ligaments, while the posterior part of the fundus was separated from the posterior abdominal wall, and therefore from the bodies of the upper lumbar vertebræ, by a wedge-shaped space, which became narrower towards the promontory of the sacrum. This space contained coils of intestine filled with gas, which must have acted like an elastic pillow in preventing any direct pressure upon the upper part of the posterior abdominal wall by the distended uterus. That the increase of the general pressure in the abdominal cavity, which the enlargement of the uterus must necessarily bring about, cannot give rise to the stasis in the renal veins and to the pathological alterations in the renal parenchyma of which we are speaking, is proved by the well-known fact that tumors due to degeneration of one or both ovaries, and much larger in size than the pregnant uterus, are occasionally developed in the abdominal cavity, where, however, they never give rise to the renal affection in question. It is true that the enormous increase of the intra-abdominal pressure may occasion diminished secretion of urine, but it never produces albuminuria. Nor must I omit to mention that the renal disease also occurs in those pregnant women in whom the position of the uterus unconditionally excludes any possibility of direct pressure upon the renal veins, namely, in women with excessively developed pendulous abdomens. I may refer to a case published by my colleague Litzmann, in the Deutsche Klinik. No. 26. 1852.

---

[1] Die Pathologie und Therapie der Nierenkrankheiten. 2te Auflage. 1870. S. 62.

[2] My colleague *Heller* has been kind enough at my request to closely follow the course of the left renal vein in a great number of post-mortem examinations. He ascertained that this vessel in the majority of cases crosses the vertebral column above the second lumbar vertebra. It lay very often in front of the intervertebral substance between the first and second lumbar vertebræ, occasionally in front of the body of the first, and was often concealed by the body of the pancreas.

In the endeavor to establish the theory of venous stasis as the cause of the renal affection of pregnancy, great stress has been laid upon the fact that the albuminuria ceases at once as soon as the pregnancy is over, or, in other words, coincidently with the removal of the assumed compression of the renal veins. If we bear in mind, however, that with the emptying of the uterus by the birth of the child a very great change takes place in the distribution of the blood to the abdominal organs, and in the blood-pressure in their vessels—a change, in fact, in the tension throughout the entire aortic system—we will be compelled to admit that these changes may well be sufficient to remove at once an inflammatory hyperæmia of the kidneys, and make the other inflammatory alterations in these organs pursue as rapid a retrograde course, as does cholera nephritis in the cases that recover. We know, both from physiological experience and from experiment, how promptly the kidneys react against fluctuations in the blood-pressure. In reality, the cessation of the renal affection at the termination of pregnancy proves no more than that the pregnancy was the cause of the renal disease.

Moreover, when the nephritis of pregnancy has lasted a considerable length of time, this rapid recovery is by no means so absolutely certain as it would have to be according to the obstructive theory, and as it really is in cases that are due to temporary venous stasis from demonstrable causes. My thanks are due to my colleague Bockendahl for the following case:

Case XIV. A young woman was attacked with albuminuria and anasarca in the course of her first pregnancy, in the year 1870. During delivery she had slight eclamptic attacks. Labor took place on November 18th, a few weeks before the proper time. Before the end of the month the albumen had entirely disappeared from the urine; the child lived.

Her second confinement ran a favorable course without any contre-temps. In 1873 she conceived for the third time while she was residing in a malarious district; during her pregnancy she was attacked with intermittent fever, and suffered much from vomiting. The ague hung about her until the end of the summer, in spite of a change of residence into a district free from malaria. She was seized with eclampsia during the night of October 29th, and while in a comatose condition, after the fit, was delivered of a fœtus of the fifth month. Consciousness did not return until the 4th of November, the patient, as in commotio cerebri, recovering recollection first of circumstances that had happened long before. The first urine passed after delivery contained so much albumen that it coagulated into a stiff, gelatinous mass when boiled. By the 27th of November a considerable reduction in the quantity of albumen contained in the urine was noticeable. The albuminuria, however, continued with a fluctuating percentage until the beginning of February, 1874.

In this case, it is true, we must admit that it was at least possible that the renal affection was dependent upon malaria and not upon the pregnancy. However, a case observed by my colleague, Litzmann,[1] proves unequivocally that the symptoms of the nephritis of pregnancy do not by any means always subside with the emptying of the uterus, but may continue for months after delivery. In a later work,[1] Litzmann reports several cases in which the first severe uræmic symptoms appeared a long time after parturition, and adds: "Altogether I am more inclined than I once was to the opinion that the disease which begins in the kidneys during pregnancy, not unfrequently persists as a chronic affection after delivery." For evidence as to the correctness of this opinion, he refers to the histories and the post-mortem appearances in several cases reported by himself. As evidence in favor of the theory of obstructive stasis, Rosenstein adduces the fact that at the autopsies on fatal cases of puerperal nephritis, atrophy of the kidneys has been exceedingly rarely met with. This argument might, it is true, seem applicable from the standpoint still held by Rosenstein, who assumed for the so-called *morbus Brightii* a regular development through three stages, but it clashes with the general experience that what Bright described as atrophy of the kidneys is never developed in the course of a few months.

---

[1] Deutsche Klinik. 1852. No. 31.

While we must consequently reject unconditionally the mechanical explanation of the mode of origin of the nephritis of pregnancy, because no tenable arguments can be adduced in favor of this theory, we are unfortunately compelled on the other hand to admit that all the other explanations advanced are equally untenable. Frerichs compared the condition of the blood in pregnancy to that "crasis" which underlies the cachectic condition; the renal disease, however, frequently attacks the most blooming and robust woman, whom it would be absurd to call cachectic. English writers have endeavored to refer the disease of the kidneys to the additional work thrown upon them during pregnancy, when they were compelled to eliminate excessively large quantities of excrementitious matters. Others have thought that some deleterious substances might be contained in the blood of pregnant women, which are capable of causing disease of the kidneys, and at the same time convulsions. These substances, however, have not yet been found. That there must be some special cause for the renal affection of pregnancy is proved by the fact, that changes analogous to those in the kidneys are in rare cases developed in the liver also during pregnancy. For these, too, the pressure of the pregnant uterus has been brought forward as the cause, but with as little success as in the case of the kidneys. For the present there is nothing left for us but to record the fact that parenchymatous inflammations of the kidneys and the liver may be developed during pregnancy, and to confess that we do not know what causes them.

---

[1] Deutsche Klinik. 1855.

Special Etiology.

The nephritis makes its appearance usually in the last months of pregnancy, and attacks primiparæ more frequently than women who have already borne children.

Even these facts which we have learned by experience have been made to lend support to the theory of pressure upon the renal vessels which I have contended against. The importance which has been ascribed to them, however, is nullified by the antagonistic facts, that nephritis may occur even in the first half of pregnancy, and that, as happened in a case reported by Rayer, it may be developed for the first time during the seventeenth pregnancy in a woman who had previously passed through sixteen perfectly regular and uncomplicated pregnancies.

Moreover, the fact that relapses of the nephritis are not unfrequently observed in recurrent pregnancies, is certainly worthy of notice. This appears to indicate the existence of some special individual predisposition; but, so far as I know, no cause for this predisposition has as yet been discovered in the constitutional conditions of the affected individuals. It may not make itself felt in every pregnancy. There are some cases on record which prove that a nephritis may complicate one pregnancy and be absent in the next, but may recur again in the third or the fourth.

In multiple pregnancy the predisposition to nephritis is so marked that Litzmann, as he has personally stated to me, in doubtful cases decides against a twin gestation, when the urine contains no albumen. This fact, too, has been made use of by the advocates of the obstructed circulation theory. Unfortunately for them, in cases of extreme distention of the uterus from hydramnios, according to Litzmann's experience, albuminuria does not occur. Twin pregnancy, therefore, can only possess the influence mentioned because the unknown cause of puerperal nephritis is then more powerful. Nephritis occurs more frequently in younger women than in those of a more advanced age. This is unquestionably due to the fact that primiparæ are affected more frequently than multiparæ. The state of the general nutrition in pregnant women has no influence on the development of nephritis. I have observed it with special frequency in robust plethoric women. Occupation and habits of life are without influence on its production. It attacks the pregnant women in every class of life, those who are encompassed by the brightest and most luxurious surroundings, as well as those who live in the greatest misery.

Nephritis is a frequent complication of pregnancy. I do not think that sufficient reliable statistics have been collected to enable a decision to be formed as to the percentage of pregnant women who are attacked by this renal affection. Whether or not this complication varies in frequency in different countries and districts, is a question I cannot answer.

Braun states that in Vienna 44 cases of eclampsia occurred in 24,000 confinements. If, now, we assume that nephritis was the cause of the eclampsia in all these cases, and further admit Rosenstein's conclusion that in one-fourth of all the cases of nephritis eclamptic attacks occur, we find as the result that one case of nephritis occurs in about 136 cases of pregnancy.

Course of the Disease.

The course pursued by the acute parenchymatous nephritis of pregnancy differs in some respects from that followed by acute renal inflammation due to other causes. In the first place, complications with other pathological processes are, as a rule, absent. The renal affection attacks individuals who were previously quite healthy, and we must admit, therefore, that the pregnant state constitutes one of the recognized disturbances or functional disorders which are often associated with this disease as determining causes.

The nephritis which is really due to the pregnancy itself, and not to any other accidentally present etiological influence, is invariably developed, so far as my own observations go, without any very striking disturbances of the general health, without febrile symptoms, and especially without any local symptoms. In the great majority of the cases the first symptom which attracts attention to the kidneys is the dropsy. This renal dropsy is distinguished from the œdema of the lower extremities, that not infrequently occurs in pregnant women whose kidneys are healthy, by the fact that it is usually not confined to the lower extremities and external genitals, but affects at the same time the hands and face as well. If the functional performances of the kidney be now investigated, the excretion will be found to be quantitatively diminished and the urine often excessively albuminous. I have never found so large a percentage of albumen in the acute nephritis due to other causes as I have repeatedly found in the nephritis of pregnancy. The urine of the latter, moreover, unlike that of the former, rarely contains blood in any quantity. Still, I have seen one case of true hemorrhagic nephritis during pregnancy which, however, pursued as favorable and rapid a course as do most of the others. My colleague, Bockendahl, has also met with a similar case. The formed elements found in the sediment of the urine (with the exception of the red blood-corpuscles) are exactly the same as in the other forms of acute nephritis. The number of hyaline casts varies greatly in the different cases; ordinarily, they are not very abundant. In many cases of the nephritis of pregnancy no œdema occurs at all; in such cases the patient feels herself quite well, no medical advice is sought, and the urine, consequently, is not examined. The renal affection then remains latent, and perhaps often enough subsides again after delivery as quietly and imperceptibly as it was developed. In some of these cases, however, either before the termination of the pregnancy, or at the commencement of labor at the natural term, or, more rarely, after the labor has been successfully terminated, those perilous attacks occur which have lent such terrible significance to the renal affection of pregnancy. Again, the nephritis of pregnancy, whether it be accompanied by œdema or not, differs from the other forms of acute nephritis by the greater frequency and violence of the acute uræmic symptoms which it causes, of the epileptiform convulsions (eclampsia gravidarum parturientium et puerperarum), the amaurosis, and the maniacal excitement.

That acute uræmia occurs more frequently in the nephritis of pregnancy than in the other forms of this renal disease, will be admitted even by those who, like myself, are not inclined to regard every case of eclampsia or mania occurring during parturition as of uræmic origin, even although the urine, after the outbreak of the symptoms, contains some albumen. Rosenstein collated the statistics given by different writers, and estimates the relative frequency of eclampsia, as compared with the albuminuria of pregnancy, at something over twenty-five per cent. The same author, from a similar comparison of the accounts of different observers, estimates the mortality among the mothers thus affected at about thirty per cent.

If the patient survives the violent convulsive attacks which, according to Litzmann's observations, may exceed thirty in number, and finally recovers consciousness after the usually protracted coma, she has, as a rule, no recollection at all of the delivery which has meanwhile taken place, for the coma that intervenes between the several attacks is usually permanent. Litzmann observed one case in which recovery took place after more than thirty paroxysms; but, on recovering consciousness, this patient had lost all recollection of the occurrences of the last half-year of her life. The same thing happened in the case of Bockendahl's described above. The mortality of the infants born under these untoward conditions is still greater than that of the eclamptic mothers. Scanzoni estimates it at forty-four per cent. In those cases in which the gestation, the parturition, and the puerperal period are safely tided over without uræmic symptoms, recovery from the renal disease takes place, as a rule, quickly and completely, as we have already stated. Nevertheless, Litzmann's experience seems to show that the transition into a chronic renal affection is more common after the nephritis of pregnancy than after any of the other forms of acute nephritis.

Pathological Appearances.

The autopsies made upon the cases which end fatally have revealed changes in the kidneys differing in degree, just as happens in all other forms of acute nephritis. For the danger of this renal affection is not dependent upon the degree to which the pathological changes have advanced, in the sense that the fatal termination only occurs when the disease is very far advanced, and never when it is but slight in degree. Other factors, besides the renal affection and the functional disturbances entailed by it, must be taken into account in explaining the death. I must expressly state that here as well as in the other forms of acute parenchymatous nephritis, both kidneys are affected similarly and to the same extent, except, of course, when there are accidental complications with old renal disease. I see, however, from Litzmann's[1] compilation that these complications have been observed with striking frequency. The left kidney is never alone affected, nor is it even affected to a greater degree than the right by the acute changes, as we would expect it to be according to the stasis theory.

As the entire process seems seldom or never to run so violent a course in pregnant women as in many other cases, the extreme degrees of hyperæmia culminating in actual extravasation—such as we occasionally meet with in the nephritis of scarlet, typhus, and recurrent fevers—occur only quite exceptionally in the nephritis of pregnancy.

I have already stated that two cases have been reported, one by Bockendahl and one by myself, in which the occurrence of hemorrhages into the kidneys was positively demonstrated by the symptoms. Both of these cases recovered; whether similar cases have been observed by others, or whether hemorrhagic effusions in the kidneys of pregnant women have ever been found at the post-mortem examinations, are questions that I am unable to answer. Apart, however, from hemorrhagic extravasations which are wanting too in a great many cases of scarlatinal nephritis, the pathological changes in the renal disease of pregnancy are identical in every respect with those of the other forms of acute nephritis. If we read the often quoted note which Virchow[1] added to his paper: "*Der puerperale Zustand, das Weib und die Zelle*," and which, strange to say, has been quoted both pro et contra, we will find in it the following statement: "In both organs (the kidneys and the liver)—and, indeed, it is a question whether the spleen too ought not to be included with them—analogous parenchymatous swellings, due to the absorption of a granular, cloudy, and apparently albuminous substance into the interior of the glandular cells, are found, in consequence of which the whole organ becomes enlarged and diminished in consistency, and after its capsule is removed appears flabby. Very often these changes present an inflammatory character, and they may then be described as parenchymatous nephritis or hepatitis. In other cases the inflammatory nature of the changes is less apparent, and we must then be content to speak of them as an albuminous infiltration. In either case the secretory function of the organ suffers, and further research is required to decide which of the two exerts the greater influence."

---

[1] Deutsche Klinik, 1855.

There can be no doubt that the author has here described simply different degrees of the same process; the nature and the genesis of the changes are identical. Unfortunately, we fail to find in Virchow's description any account of the appearances in the interstitial connective tissue in the nephritis of pregnancy. I am not aware that any attention has been paid to the intertubular substance even in the more recent studies on such kidneys.

When we study carefully the descriptions of the macroscopic appearances of kidneys removed from the bodies of lying-in women who have died of eclampsia, a number of which were published by Litzmann in the Deutsche Klinik for 1855, we find that they correspond exactly with the appearances which, as we saw in the preceding chapter, are presented by acute renal inflammations due to other causes—for instance, by scarlatinal nephritis that has run a protracted course. The kidneys are larger and heavier than normal from thickening of the cortical substance. The cortex is anæmic, of a pale yellowish color and of swashy consistency. What resemblance is there here to the very vascular, dark colored, and usually firm and compact kidneys that we find when venous stasis has existed? It is unnecessary to add anything concerning the changes in the other organs of the body that are found at the autopsy. I will only mention that dropsical effusions are seldom altogether absent. Thus Braun found œdema in thirty-nine out of forty-four cases of eclampsia.

---

[1] Gesammelte Abhandlungen. p. 778.

Analysis of Individual Symptoms.

The functional disturbances of the kidneys in the nephritis of pregnancy make their appearance in the same way as in the other forms of acute parenchymatous nephritis. I do not know that complete arrest of the renal secretion ever takes place in the former, but I have already stated that absolute suppression of urine is seldom met with even in the other forms of acute renal inflammation, and that, except after cholera, I have myself only encountered it after scarlet fever and diphtheria. As the renal affection usually commences without any special symptoms, without pain and without fever, and as certain symptoms which might excite suspicion—such as, for instance, the frequent desire to pass water—are readily attributed by the patient to the pregnancy, accurate analyses of the urine during the first stage of the disease are to a certain extent wanting.

When it has advanced far enough to cause œdema, we find the excretion of urine diminished; its specific gravity is increased, for the same reasons as in the other forms of acute nephritis, and it contains a great deal of albumen, casts, etc. On the advent of convalescence, which usually does not set in until after the birth of the child, this diminution gives place to an abnormal increase of the excretion, the urine presents an abnormally low specific gravity, and the albumen disappears. It is unnecessary for me to repeat here all that I have already said concerning the excretion of the urine in acute nephritis, which applies just as well to the nephritis of pregnancy. The only difference is that in the latter the transition to a state of convalescence, *i. e.*, the re-establishment of the normal conditions of secretion, takes place, as a rule, at a fixed period, namely, at the termination of the pregnancy.

In order, however, to demonstrate by positive proof the correctness of the opinion that the excretion of urine follows exactly the same course in the nephritis of pregnancy as in the other forms of acute parenchymatous inflammations of the kidneys, I will briefly detail here results of some examinations of the urine of a pregnant woman, which I recently had the opportunity of making.

Case XV.—A young lady of robust build, full figure, and blooming color, who had had no previous illness, was married in the early part of 1874, and conceived shortly after her marriage.

Late in autumn her friends observed that her face was somewhat puffed. Her hands and feet shortly became anasarcous. In other respects the patient felt quite well, and she did not think it necessary to consult a doctor.

Some days before my investigations began, her friends were struck by a remarkable change in the disposition of the patient, by a singular apathy and lack of resolution; next there ensued, as it appears, temporary disturbances of the visual organs. All this did not prevent the patient from going out as she had been in the habit of doing. During one of her walks she was seized in the street with eclamptic convulsions, which, however, seemed to have been of slight intensity, and were not repeated. Consciousness was quickly recovered.

Two days subsequently, on December 22d, I received the urine passed during the preceding twenty-four hours. The total quantity was 750 c.c., sp. gr. 1018, and it contained 0.296 per cent. of albumen and 1.9 per cent. of urea. The quantity of urine passed on the succeeding days was less, but unfortunately the entire quantity could not be collected every day. The smallest total occurred on December 25th, when only 470 c.c. were passed; its sp. gr. was 1024, and it contained 0.653 per cent. albumen, and 2.2 per cent. urea. The highest sp. gr.—1028—was attained on December 26th, and the largest percentage of albumen = 1.568 per cent., on December 28th.

In the commencement of January, the secretion of urine began to be abundant. At the same time the specific gravity fell below the normal, and the percentage of albumen and urea diminished, although the total amount of urea excreted was considerably increased, as was also to a less extent that of the albumen. During the first week that the case was under observation, five analyses were made, and I found that on the average, 590 c.c. of urine were passed daily, containing 4 grammes of albumen and 13.4 grammes of urea. while the following figures, which were estimated from seventeen analyses, would represent the average quantities during the first three weeks of January:

Total quantity of urine passed daily	1645 c.c.
Total quantity of albumen passed daily	9.15 grms.
Total quantity of urea passed daily	22.65 grms.

In the meantime the œdema completely subsided, and from the time that the polyuria began (on the 5th of January 2110 c.c. were passed) the young lady enjoyed excellent health. During the night of the 3d of February she was delivered, after a pretty long but uncomplicated labor, of a strong, living boy, who throve excellently on his mother's breast. Even at the present time, however, while this work is in press (at the end of May), the urine of the young mother still contains albumen and a few casts. Consequently this is another case which proves that the albuminuria by no means invariably ceases at the termination of pregnancy.

Such conditions of the urinary secretion as were found in the above case, and especially so high a percentage of albumen, do not occur in cases of obstructive congestion of the kidneys. The urine on one of the days on which it was analyzed contained, as above mentioned, 1.568 per cent. of albumen. Spiegelberg[1] drew off by catheter, from the bladder of an eclamptic patient who was in the throes of labor, urine that contained even 4.78 per cent. of albumen. Rosenstein admits that the urine in the nephritis of pregnancy does not present simply the characters due to the altered condition of the circulation, but bears the impress also of the altered constitution of the blood, of the marked hydræmia. But the patients with heart-disease who suffer from albuminuria are also hydræmic and also dropsical, like the pregnant women who are suffering from renal disease, and yet they never pass urine containing so large a percentage of albumen.

Unfortunately, I have neglected in my examinations to compare the percentage of albumen contained in the urine passed by day with that passed by night, during the nephritis of pregnancy. If the obstruction theory were well founded, the diurnal urine which is excreted while the body is in the upright position should contain less albumen than the nocturnal urine which is excreted in the horizontal position, because on account of the influence of gravitation the assumed pressure upon the renal veins would necessarily be less in the upright posture.

The extension of the dropsy in pregnant women who are attacked with the renal disease is occasionally affected by those disturbances of the circulation in the vessels of the lower limbs and the external genitalia which are entailed by pregnancy. It happens even more frequently than in other renal affections that these parts only are swollen, while the rest of the body remains free from anasarca. With this exception, the dropsy follows the same course as in other diseases of the kidneys; perhaps it more rarely attains an extreme degree because, as I am inclined to assume from my own limited experience, extreme dropsy (or the coincident extreme hydræmia) seems to cause death of the fœtus and its premature delivery, and with the latter the dropsy is brought to an end. On the other hand, dropsy is seldom altogether absent. The subjects of puerperal nephritis are unquestionably more exposed to the danger of uræmic attacks than the subjects of other renal diseases. At the same time I am far from thinking that every case of eclampsia must be ascribed to disease of the kidneys and uræmia, nor even every case in which albumen and a few casts are found in the urine after the attack. I have already mentioned a case of this sort from Litzmann's Clinic, in which, after death, the kidneys were found to be perfectly healthy, although during life the urine contained albumen. Albumen may pass into the urine in consequence of the violent disturbances of the circulation caused by the convulsions, and, at the same time, as Huppert's[1] observations on epileptics proved, casts of the urinary tubules may be formed. If, now—as we know from experience—the act of parturition is alone sufficient under certain circumstances to cause attacks of eclampsia, surely this process, which exerts in so many ways a powerful influence upon the nervous system, may be accredited with the principal share in the production of that marked predisposition to uræmic seizures which characterizes the nephritis of pregnancy above all other renal affections. The influences due to the retention of urinary constituents in the blood and the tissues combine with the nervous irritation occasioned by the pains of labor to produce the attacks in question. Even this, however, by no means explains the enormous frequency of uræmia in this form of nephritis. Convulsions that are unquestionably uræmic in nature often occur before the commencement of labor, or appear for the first time after its termination during the puerperal period. I have been particularly struck by the extraordinary frequency with which that otherwise rare uræmic symptom—amaurosis—has been observed in the nephritis of pregnancy. This symptom, moreover, cannot be ascribed to the direct influence of the act of parturition, even with as much probability as can the convulsions and perhaps also the mania, since it ordinarily makes its appearance before the commencement of the actual labor-pains. Still, the influence of the act of parturition on the production of the eclamptic attacks should not by any means be disparaged. I borrow from Rosenstein the following statistics, which he has compiled from the figures given by Braun and Wieger: In 449 cases of eclampsia the convulsions occurred 121 times before the labor-pains set in, 260 times during labor, and 118 times after the birth of the child during the puerperal period. Of the cases in the last category a considerable portion might perhaps be brought into connection with after-pains. I will also mention another of Rosenstein's tables, which shows that out of 115 cases of eclampsia, in 39 the convulsions ceased entirely after the birth of the child; in 36 they recurred, but were much less severe; while only in 37 did they continue violent.

---

[1] **Archiv für Gynäkologie. Bd. 1. S. 386.**

---

[1] Virchow's Archiv. Bd. 59. Heft. 3 u. 4.

All this collected experience as to the direct bearing of the act of parturition upon the development of convulsions will not suffice, however, to explain the striking frequency and equally striking violence of the uræmic symptoms in the nephritis of pregnancy as contrasted with the other forms of acute inflammation of the kidneys, since, as the above figures show, in the large proportion of cases the act of parturition cannot be taken into account at all. There must, therefore, of necessity be still other influences at work, the nature of which might perhaps be discovered by more careful and more extensive investigations of the urine of pregnant women than have hitherto been instituted. Rosenstein endeavors to explain the occurrence of convulsions upon Traube's theory. He assumes, in the first place, that every pregnant woman is hydræmic, and makes the labor-pains and the abdominal pressure supply the increased pressure in the aortic system, which is exacted by Traube. But then all pregnant women are not hydræmic, and even in those who are attacked with nephritis and eclampsia the blood is not necessarily very watery. Some eclamptic patients maintain a blooming appearance, and present no traces of anasarca. It may be admitted that during the labor-pains the uterine arteries are compressed, and that a general increase of the tension in the arterial system necessarily results therefrom, but it has not yet been proved that the tension in the aortic system is increased by the action of the abdominal pressure. That the influences just mentioned increase the pressure in the venous system is certainly demonstrated by the bluish color of the face during the expulsive pains of labor. But this would indicate a diminution rather than an increase of the tension in the aortic system. Litzmann very often noticed a remarkable slowing of the pulse before the outbreak of eclamptic convulsions, and I have frequently observed the same thing in the other forms of parenchymatous nephritis, in the chronic as well as in the acute. Uræmic symptoms, however, did not invariably follow this symptom. The chief points to be borne in mind are that the eclamptic attacks and other uræmic symptoms are so frequently entirely independent of parturition, and that for the development of the eclamptic attacks no particular grade of the renal affection is demanded. In reference to this latter point, Virchow[1] says: "And so far as the changes in the kidney are concerned, I have seen at least as marked departures from the normal state in puerperal women who had had no eclampsia and even no convulsive attacks, as in those who had been truly eclamptic. Even if it would become possible to establish a constant connection between eclampsia and uræmia, we should always be compelled to assume some special predisposition in the nervous system, and my object in the foregoing remarks was to demonstrate this during pregnancy."

One peculiar symptom which I do not recollect having observed in other forms of acute nephritis is mentioned by Litzmann as of frequent occurrence in the nephritis of pregnancy, namely, attacks of neuralgic pain in the region of the kidneys. These attacks were always preceded by a diminution of the urinary secretion, and upon the subsidence of the paroxysm the excretion again became abundant. It is perhaps due to a temporary compression of the ureter by the gravid uterus.

---

[1] L. c. S. 778.

Prognosis.

The prognosis of the acute parenchymatous nephritis of pregnancy is less favorable than that of many other forms of acute renal inflammation, solely in consequence of this decided predisposition to uræmia. I have already stated that according to Rosenstein's statistics, eclampsia occurs in about one-fourth of all the cases, and that about thirty per cent. of the eclamptic patients die. The danger to life, according to Litzmann's experience, is due more to the violence than to the frequency of the convulsive attacks. In those cases, however, that escape this peril, the prognosis is rather more favorable than in the cases of acute nephritis of other origin. In most cases convalescence sets in immediately after delivery. I do not know that women who are suffering from the renal disease, apart from the risks of uræmia, are threatened with greater danger during the puerperal period than previously healthy women. It is certainly surprising that the former do not present a greater proclivity to inflammatory affections—to peritonitis, for instance, than the latter, since diffuse affections of the kidneys are in general very apt to cause inflammatory processes in the serous membranes.

I have already stated that the nephritis frequently relapses in subsequent pregnancies, especially when they follow one another in rapid succession, and that this form of acute inflammation of the kidneys, in Litzmann's opinion, exhibits a more marked tendency to develop into a chronic kidney disease, than the other forms. It is also known that premature deliveries are frequent in pregnant women with diseased kidneys. Perhaps, as I have already remarked, this is in many instances due to the fact that the fœtus cannot derive sufficient nourishment from the excessively hydræmic blood of the mother, and therefore dies. Even well-developed, strong children, however, frequently die either during or soon after delivery at full term, when the mothers have been attacked with eclampsia during labor. The cause of this remarkable mortality is not known with certainty. Does the fœtus participate in the uræmic poisoning of its mother? An affirmative answer to this question must necessarily appear justifiable to all those who regard the chemical pollution of the blood as the cause of the uræmic attacks in all, or even in a part of the cases. Litzmann[1] records an observation that furnishes a positive foundation for this opinion. Panum obtained in the ordinary way a pretty large number of crystals presenting forms similar to those of nitrate of urea, from the sanguineo-serous fluid found in the pleural cavities of a child whose mother was attacked with eclamptic convulsions during labor. The child died twelve hours after its birth.

Diagnosis.

With regard to the diagnosis of the nephritis of pregnancy, I may refer to what has been said above concerning the diagnosis of acute parenchymatous nephritis in general. In consequence of the presence of the notorious etiological influence it will be even more easily arrived at than in cases of nephritis due to other causes. It is of course necessary that the pregnancy itself should be diagnosticated; and this is all the more easy since the nephritis, as a rule, is not developed before the latter half of the pregnancy. We have only to avoid confounding it with any chronic affection of the kidney, which may have existed before impregnation.

Treatment.

The treatment must, in my opinion, be conducted upon exactly the same principles as those laid down by me for the treatment of acute parenchymatous inflammation. The pregnancy does not appear to me to counter-indicate the production of an energetic diaphoresis, and this seems all the more indicated because the renal affection nearly always runs its course without febrile movement. I have already stated that my treatment of the uræmic condition in other renal diseases was determined by the experience I had derived from cases of eclampsia in pregnant women. The treatment of eclampsia occurring during labor should be guided by the experience which has taught us that the dangerous accidents cease entirely in nearly one-third of all the cases as soon as the uterus is emptied: the labor should be terminated by artificial means as soon as they can be employed without danger to the mother or child.

---

[1] Monatsschrift für Geburtskunde und Frauenkrankheiten. Bd. 11. S. 424.

b. Chronic Parenchymatous Inflammation of the Kidney.

This is the second stage of the Bright's disease of most writers—the non-desquamative nephritis of Johnson, the néphrite parenchymateuse profonde ou grave of Lecorché.

Samuel Wilks[1] was the first to prove, and he did so in the clearest possible manner from the ample clinical and pathological materials at his command, that the condition of the kidney, of which we are now talking and which he described as "the large white kidney," ought not to be regarded as the precursory stage of that atrophic process which the German pathologists had proclaimed as the ultimate stage of every diffuse inflammation of the kidneys, as the third stage of their morbus Brightii. He taxes Frerichs with his failure to support his theory as to the succession of the three stages of Bright's disease assumed by him, with a single case which began with the symptoms of acute nephritis; in other words, with an acute hydrops, and in which, after it had lasted for years, small contracted kidneys had been found. He adds that his own experience has not furnished him with a single case of this sort. Wilks does not distinguish between the acute and chronic forms of parenchymatous nephritis. The clinical picture which he sketches would therefore apply only to those few cases which really have an acute commencement; my own observations related in the last chapter show that this sometimes occurs. To the great majority of the cases his description is not applicable in the respect that the initial fever and the hæmaturia are, as a rule, entirely wanting. Todd recognized the error in the view held by his countryman.

---

[1] Cases of Bright's disease with remarks. By *S. Wilks*, M.D., Lond. Guy's Hosp. Rep. 1853.

In Germany the work of Samuel Wilks seems to have received little or no notice or recognition. At all events, it exercised no influence upon the descriptions of the diffuse renal disease that appeared in our hand-books of clinical medicine.

Among the French, Kelsch was the most decided in admitting the individuality of the chronic renal affection which Wilks described as "large white kidney." He denies its inflammatory nature, however, and asserts that Wilks did so too, which is incorrect, for in direct contradiction to Kelsch's views Wilks describes the swelling of the large white kidney as the product of an inflammation which, starting from the urinary tubules, is in the early stages capable of undergoing complete resolution. He likens this process to a bronchitis. In both instances the inflammation may extend from the tubes first affected to the tissues around them; in the kidneys the entire tissue may be saturated with inflammatory exudation, while the bronchitis may terminate in broncho-pneumonia.

Kelsch denies that the affection has an acute initial stage, and prefers to regard it as an anæmic necrosis of the epithelial cells associated with swelling, but not implicating in the least the rest of the renal tissues. He claims that it attacks only persons whose general nutrition is reduced (phthisical, scrofulous, and syphilitic subjects); that the general anæmia causes ischæmia of the kidneys, and in this way conduces to the degeneration of the epithelium. In what follows I think I shall be able to prove the incorrectness of this view from an etiological as well as from a pathological and a clinical standpoint.

Etiology.

Chronic parenchymatous nephritis is developed from acute inflammation of the kidneys in a few cases, but in the great majority of the cases it progresses in an insidious manner from the very beginning.

I have already remarked that in the different varieties of acute parenchymatous nephritis the tendency to develop into a chronic affection is very variable. I know of no case of nephritis after cholera or diphtheria which became chronic ; but, on the other hand, in the nephritis both of scarlet fever and of pregnancy the transition into a chronic renal disease has undoubtedly been observed. Whether the inflammation of the kidneys that follows small-pox and typhoid disease has been observed to follow the same course I am unable to say, but I can positively assert that the inflammation of the kidneys which is excited by exposure to cold may develop into a chronic affection. With regard to the cases of acute nephritis that are secondary to excessive suppuration of the cellular tissue, we may even assert that they possess a decided tendency to run a chronic course.

I am unable to say on what this difference in the different cases depends; to explain why, of affections that differ only in their causes, the anatomical lesions and the symptoms being identical, one should present a greater tendency to develop into a chronic disease than another. Nevertheless, the fact in itself compels us to assume that the obstinate persistence of the affection, and its consequent development to higher grades of pathological change, must depend either upon the intensity of the action of the original exciting cause, or upon a protracted working of the same cause.

This view is supported, at least in part, by the observations that have been collected with reference to the etiology of the form of parenchymatous nephritis, which, from the very beginning, pursues a chronic and insidious course. These causes are essentially identical with some of the known causes of the acute affection of the kidneys.

The parenchymatous inflammations of the kidneys which from the very commencement pursue a chronic course are developed with extraordinary frequency during the course of affections that are accompanied by persistent suppuration, such as diseases of the bones and joints, the more severe forms of inveterate syphilis, phthisical, ulcerative destruction of the lungs, etc. These I hold to be the most common sources of the renal affection we are discussing, and I cannot rid myself of the notion that something is developed in these collections of pus which is taken up into the blood by absorption, and excreted by the kidneys, and which during its excretion excites an inflammation of these excretory organs. When there is acute suppuration of the cellular tissue, the sudden excretion of a large quantity of this "something" causes an intense inflammation in the kidneys, which, however, pursues a rapid course, and disappears as soon as the source from which the agent is supplied is cut off by a thorough evacuation of the pus, and by preventing it from forming afresh. On the other hand, a chronic nephritis with fatal issue may ensue if the treatment pursued be insufficient to secure this end. In chronic suppurations this unknown "something" is constantly formed and taken up into the blood in small quantities, and slowly and insidiously causes the same process in the kidneys which in the other case is ushered in by a violent initiatory stage.

Experience has shown that it is wrong to attribute, as has of late years been the fashion, every case of albuminuria that is secondary to chronic disease of the joints and bones, to inveterate syphilis, pulmonary consumption, chronic ulcers, etc., to amyloid degeneration of the blood-vessels of the kidneys. Clinical observation and anatomical examinations have proven that a large portion of these cases may be credited to the account of chronic nephritis. I must lay stress here, however, on the fact that chronic parenchymatous inflammation of the kidneys by no means excludes the possibility of an amyloid degeneration of their blood-vessels; on the contrary, the two affections are very frequently found combined in the same subject when some chronic suppurative process has preceded the renal disease.

Just as chronic suppurations cause a chronic inflammation of the kidneys by means of the persistent action of small quantities of the noxious agent, while the sudden action of a larger quantity of the same agent produces acute nephritis, so the persistent influence of continued exposure to cold and moisture brings about the same chronic renal affection, while a severe cold caught by sudden exposure is followed by acute nephritis. Almost all writers, among the causes of chronic Bright's disease, mention living in cold and wet habitations, occupations in which the body is frequently wetted and chilled through, etc. My own experience does not enable me either to confirm or to contradict these statements.

I may mention marsh miasm as a decidedly frequent exciting cause of chronic parenchymatous nephritis, next to chronic suppuration perhaps the most frequent. A not inconsiderable number of cases have been admitted into the hospital in this city during the last ten years, from the marshy districts bordering on the Elbe and the North Sea in Sleswick and Holstein, in whom this grave renal disease had developed after long-continued intermittent fevers. I have also received many such cases from the coasts of the Baltic and from the intervening parts of the province, where intermittents, as a rule, are rare. As to the way and manner in which malaria causes inflammation of the kidneys, I have not been able to form any decided opinion. At first I thought it possible that, in consequence of the extreme disturbances of the circulation connected with the paroxysms of high fever, or in consequence of the excessive elevation of the temperature observed in many cases, some lesion of the renal vessels might be produced, the frequent repetition of which might cause the development of a state of inflammation. I was confirmed in this opinion by the observation of two cases of ordinary tertian intermittent fever, in which albuminous urine was passed during the paroxysms, but in which the urine lost the abnormal ingredient when the attacks were stopped by quinine. Since then, moreover, I have met with a case which appeared to demonstrate that malaria may under certain circumstances produce parenchymatous nephritis without the intervention of febrile paroxysms, just as it sometimes causes splenic tumors without preceding attacks of fever. The patient was an engineer, thirty years of age, and from this district, who had never suffered from any sickness before the year 1873, when he undertook the erection of a dyke at Jahdebusen. A few months after his arrival there he noticed an œdematous swelling of his lower limbs. The dropsy rapidly increased, and in February, 1874, when he was admitted into the hospital here, his whole body was anasarcous. He was still in the hospital in March, 1875; the swelling had then disappeared, but his urine still contained a large quantity of albumen.

The older English writers regarded the misuse of mercury as a cause of albuminuria and disease of the kidneys. Rayer denied that mercury had any such effect. Kussmaul[1] relates a case of mercurialismus affecting a mirror-silverer thirty-one years of age, whose urine contained albumen as well as mercury. The quantity of albumen contained in the urine diminished as the erethismus mercurialis improved, and disappeared entirely as soon as recovery was complete. Kussmaul's own experience and that of others satisfied him that hydrargyrosis is occasionally accompanied by albuminuria, but he adds: "Whenever serious disease of the kidneys has been found at the autopsies of mirror-silverers who had previously suffered from mercurialism, it has always been possible to connect the disease with a coexistent pulmonary tuberculosis."[2]

My own experience has not enabled me to form a decided opinion as to the possibility of parenchymatous nephritis being caused by the action of mercury. In the course of years I have probably treated two thousand cases of constitutional syphilis with mercury, and have been by no means timid in the employment of this indispensable remedy. Among the syphilitic cases thus treated, some, it is true, have died of chronic parenchymatous nephritis; but I have also seen persons die of the same sequel of inveterate syphilis who were brought to the hospital from distant country districts in a most miserable plight, and who had not taken an atom of mercury either before or after their admission.

According to a summary of the results of the mercurial treatment of syphilis in our hospital, published a few years ago by Dr. Schorer, it appears that the average duration of the treatment was about seventy days, but it was often protracted considerably beyond this period. Allowing for the interruptions rendered necessary by intercurrent affections of the mouth, on an average about 300 grammes (not quite ten ounces) of gray mercurial ointment were rubbed into each patient. In persons who were not already suffering from albuminuria, I have never known these large doses of mercury to cause the slightest disturbance of the renal functions. The following case, however, made me for a time suspicious of the innocuousness of the excessive employment of the inunction cure with mercurial ointment, although it can by no means be regarded as positive proof of an injurious action of the same on the kidneys. I shall relate the case at rather greater length than would otherwise be appropriate in this place, because it contains a number of interesting points.

---

[1] Untersuchungen über den constitutionellen Mercurialismus, etc. Würzburg, 1861. S. 167.

[2] L. c. S. 326.

Case XVI.—L. S., a maid-servant, from Dithmarschen, and at that time 22 years of age, was admitted for the first time into the Academical Hospital at Kiel, on the 17th of May, 1857. She was suffering from a serpiginous ulcerative destruction of the skin of the neck and face, which had existed for several years and had already destroyed a considerable part of the alæ and tip of the nose. There was also an ulcer with exuberant granulations on the hard palate, and a lachrymal fistula on the left side. The diagnosis was morbus Dithmarsicus (hereditary syphilis). The treatment consisted in the employment of Zittman's decoction; later on, cod-liver oil was given, and during the summer she took sea-baths. On December 29th the patient was discharged completely cured, even the lachrymal fistula having closed of itself without operative interference. In 1858 she was admitted again for a relapse, but I could not find the records of the symptoms and treatment. She was admitted a third time into the hospital on June 13, 1862. Some ulcers had developed again on the site of the old cicatrix, and some nodules arranged in circular groups had appeared in the skin of the right cheek. The patient was emaciated, and presented a very cachectic appearance. For this reason the mercurial treatment was deemed inadvisable, and in its stead the patient was put upon iodide of potassium and the bitter-wood infusions, together with local applications. As this treatment proved fruitless, an attempt was made to effect a cure by syphilization. In the course of about three months nearly 250 chancres were produced by inoculation on the thighs and abdomen. Notwithstanding this, the patient recovered her strength. The sores on the nose and face, however, did not heal. Finally, complete recovery was brought about by a mercurial course, of which unfortunately no particulars are recorded. She was discharged on November 2, 1863. A fourth relapse took place in February, 1867. Zittman's decoction, though given for a long time, and iodide of potassium, caused no improvement; but she convalesced under a *thirst cure*, and was discharged on January 1, 1868.

After her recovery the patient entered the hospital service as a nurse, and remained healthy until May, 1870. Paralysis then set in very gradually, affecting first the lower, and then the upper extremities; she finally became completely helpless, and for a long time had to be fed. Treatment by inunction was once more resorted to, combined with the internal use of iodide of potassium, and was persisted in, as her mouth never became affected, up to the date of her complete recovery in March, 1871. She then resumed her situation as nurse. Only a few days elapsed, however, before the paralysis returned without any febrile disturbance, affecting first the left arm, then the left leg, then the right arm, and finally the right leg. For some days the sphincters of the bladder and rectum were also paralyzed, but they soon resumed their normal functions. None of the cerebral nerves were affected. The urine contained a small quantity of albumen. Bed-sores appeared over the sacrum and shoulder-blades. In the evenings she had slight fever, and occasionally painful contractions of the flexor muscles of both thighs occurred. *The power of sensation in the paralyzed limbs was not affected.*

Ordered inunctions and iodide of potassium. Gradual improvement. By the beginning of August the bed-sores had healed, and the urine was free from albumen. The development of stomatitis made it necessary to discontinue the inunctions. The inunction treatment was continued, although with repeated interruptions rendered necessary by recurring stomatitis, until May, 1872. It was then estimated that over 2,000 grammes of mercurial ointment had been rubbed in during the course of two years. She was discharged from the hospital on the 9th of June, 1872, at the desire of the parish authorities, who were chargeable for her; she had then regained the complete use of her limbs, and her urine was free from albumen; but a few nodules had reappeared in the skin of the face. She returned again, however, on August 27th of the same year. She was emaciated, pale, and in every respect reduced since her departure.

On the right forearm, and above the left internal malleolus, two circular ulcers extending through the entire thickness of the skin were found, each about the size of a thaler; the base of the one on the leg was discolored. Numerous lupus-like nodules had developed in the neighborhood of the old cicatrices on the face. Syphilitic dactylitis of several of the phalanges and of the middle metacarpal bone of the left hand. At different parts of the body, in the subcutaneous cellular tissue, perfectly painless tumors, partly firm and partly fluctuating, and varying in size from a hazel-nut to half a hen's egg. No trace of the former paralysis, and no albumen in the urine.

Mercurial inunctions were again employed, and again the patient recovered. The old ulcers, however, healed badly, and new ones were formed by softening and breaking down at the above-mentioned subcutaneous gummy tumors. In April, 1873, the patient became feverish and began to cough. There were signs of catarrh at the apices of both lungs, and evident symptoms of consolidation first upon the left side; the inunctions were now discontinued. On the 20th May she had a moderately severe attack of hæmoptysis; during the summer the pulmonary affection advanced, and the ulcers of the skin slowly healed. In July, traces of albumen were again found in the urine. In August, the existence of vomicæ in the lungs could be demonstrated. During September, the quantity of albumen contained in the urine constantly increased, and in the beginning of November the percentage was the largest I have ever seen, the total quantity of urine being at the same time diminished. Towards the end of October, diarrhœa set in, the stools being at first bloody; this diarrhœa persisted until death. About the same time she began to swell, at first about the ankles, and she was henceforth constantly confined to bed. The hydrops anasarca rapidly extended over the entire body, and on November 10th a fluid effusion was discovered in the abdominal cavity. She died in an extreme state of exhaustion, at midday on December 8th. Directly after death, a large quantity of still perfectly fluid blood was drawn from the right jugular vein of the corpse. I take the following record of the post-mortem, which was dictated by my colleague Heller:

Great œdema of the lower extremities; numerous scars on both thighs (syphilization, fourteen years ago). Upon the back of the left hand two ulcers reaching down to bare bone, with dense infiltrated borders and lardaceous bases. A fluctuating tumor on the left forearm, containing a yellowish white fluid. In the face the bridge of the nose depressed; in the upper part of the nose a fistulous opening about the size of a pea, from which some dirty pus escapes. A sound passed through this opening reaches bare, roughened bone. In the fistulous openings, which correspond to the nares, the superior maxillary bone lies rough and bare; in the mesial line and for a considerable distance to the right, it seems to be destroyed, so that the fangs of the teeth are in some places exposed. The bony wall between the nose and the right orbital cavity perforated, the opening being about as large as a groschen. Opening surrounded by roughened edges of bone bathed in an ichorous fluid. Behind the articulations, between the atlas and base of the skull, and between the former and the epistropheus, a somewhat discolored swelling, which on section was found to be a dense, pultaceous caseous mass. The left half of the medulla oblongata flattened and broader than natural; but with this exception there was no abnormality in the central organs of the nervous system. Both lungs very small. In the apex of the right lung a small cavity with thick, pultaceous contents; in front of this cross-sections of several bronchi surrounded by caseous tissue, and connected together by dense bands of connective tissue. Posteriorly, groups of indurated, gray nodules. The lower lobe of right lung contains numerous nodules of the same kind. The surface of the upper lobe of the left lung presents depressions, caused by cicatricial bands which extend deeply into its substance; this lobe is riddled with sinuous cavities that communicate with one another. The lower lobes contain a moderate quantity of air, but a great number of closely grouped, though mostly isolated, slate-gray nodules, are dispersed through them. Heart small; coronary arteries tortuous. Valves normal. Aorta narrow. In the abdominal cavity a large quantity of fluid, which in the upper part is clear, but in the pelvis is of a deep yellow color, turbid, and deposits a sediment.

The surface of the liver connected with the parietal layer of the peritoneum by very numerous bands of connective tissue. Substance of the liver somewhat granular on section, marbled, yellow, and light brown. Spleen pretty firm; its tissue of a grayish red color on section, and sprinkled with very numerous gray nodules. Left kidney enlarged, 12½ cm. in length; the capsule in parts firmly adherent; the surface presents six irregular patches of a waxy yellow and a grayish red color. The tissue very pale on section. The cortex cloudy, spotted with yellow. The right kidney in general the same, only much paler. In the cæcum one extensive circular ulcer, 5 cm. in diameter, and numerous smaller ones. The ulcerative process extends as far as the sigmoid flexure; only a few round ulcers in the flexure itself. All the ulcers have infiltrated wall-like borders and spongy bases.

So far as I know, but few cases of parenchymatous nephritis have been published in which the affection has been observed throughout its entire course, and the characters of the urine noted from the very commencement. It would occupy too much space if I were to copy in extenso the tables obtained from our analyses. I must therefore content myself with the following account of the principal results furnished by the examinations of the urine.

The first traces of albumen were found on the 20th of July; on that day 1,100 c.c. of urine were passed, with a specific gravity 1010. The first hyaline casts were found on the 24th of August, the amount of albumen in the urine having gradually increased in the meantime.

The following table exhibits the slow increase in the percentage of albumen and in the other changes in the urine.

Date.	24 Hours' Urine. c.c.	Specific Gravity.	Albumen.		Urea.		Chlorides.	
			%	In toto.	%	In toto.	%	In toto.
Aug. 9	1200	1011	0.072	0.864				
" 21	1100	1013	0.125	1.375	1.2	13.2		
" 28	1300	1017	0.153	1.989	1.9	24.7		
Sept. 4	1300	1010	0.242	3.096	1.1	14.3		
Oct. 11	1255	1010	0.292	3.664				
" 16	1550	1012	0.774	10.997				
" 22	1985	1012	0.464	9.210				
" 26	1025	1012	1.076	11.092				
" 31	940	1015	1.184	11.129				
Nov. 3	825	1017	1.312	10.824				
" 8	135	1040	4.930*	6.656	3.4	4.59		
" 9	335	1027	2.299	7.703	4.0	13.40		
" 15	230	1027	2.568	5.906	3.0	6.90		
" 20	160	1036	2.820	4.512	1.5	2.40		
" 23	265	1032	2.216	5.872			0.132	0.352
Dec. 2	175	1030	1.964	3.437	3.3	5.77		
" 7	340	1020	1.449	4.926	5.6	19.04		

* Estimated by alcohol, 6.475.

From the time when the urinary secretion began to diminish, in the beginning of November, up to the day of her death, on December 8th, all the urine passed was collected for 36 days. This gave a mean average per diem of 350 c.c. The quantity of albumen was estimated on 30 days; on an average 6.332 grammes of albumen were discharged with the urine per diem. Unfortunately, the quantity of urea was only determined on 7 days; the average excretion of urea, according to these few examinations, was 9 grammes in the 24 hours.

As the disease progressed, the quantity of casts in the sediment increased enormously, and broad, darkly granular casts were soon added to the small hyaline casts.

The setting in of the diarrhœa in the beginning of November had an unmistakable influence on the enormous and sudden diminution in the quantity of the urine. On some days over 2,000 c.c. of fluid were discharged through the intestines. Of course it cannot be denied that some urine also may occasionally have been passed while at stool; on one occasion, however, the fluid was tested for urea, but none was found.

The character of the blood drawn from the body directly after death is also worthy of notice. The blood-serum had a specific gravity of only 1015.58, which was considerably below that of the urine passed immediately before death, and very much less than that of the urine which was passed on preceding days. The percentage of albumen in the blood-serum was 3.4 per cent., while on the 8th of November the percentage of albumen in the urine was nearly 5 per cent.

No one who reads carefully the above history will be inclined to regard this case as a proof of the influence of mercury in exciting albuminuria, as assumed by Wells and Blackall. For my own part, after this case, which I watched to the very end, I no longer hesitate to deny positively the possibility that the mercurial treatment as employed here in Germany can ever excite nephritis. The albuminuria which appeared in the spring of 1871 disappeared when the interrupted inunction treatment was recommenced; and the first symptoms of the renal disease, which finally terminated life, showed themselves several months after the use of mercury had been discontinued on account of the intercurrent phthisical affection. Before leaving this case I wish to draw attention to the anatomical explanation of the paralytic symptoms observed during life. These paralyses were evidently caused by a gummy tumor which was situated behind the articulations between the upper cervical vertebræ and pressed upon the medulla oblongata, and they disappeared when the original deposit was thickened and reduced in volume by absorption. The left half of the medulla oblongata, at the autopsy, was found still somewhat compressed and flattened.

In a considerable number of the cases of parenchymatous inflammation of the kidneys which from first to last pursue a chronic course, it is impossible from the past history of the patient to discover any sufficient cause for the development of the malady. It also occurs, and this I must particularly emphasize in contradiction to Herr Kelsch, and may follow a chronic course from the commencement, even in individuals who, previous to the outbreak of the renal affection, were in the enjoyment of blooming health and were thoroughly well nourished. I may say positively that alcoholic excesses, to which the disease is by many attributed, cannot be charged with being the cause of it. None of the cases treated by me occurred in drunkards, and in no instance have I encountered the large white kidney at the autopsies of notorious drinkers, of which I have made a not inconsiderable number during my many years' active hospital service.

It is not easy to form an estimate with regard to the frequency of chronic parenchymatous nephritis in general, since the older writers, and the Germans in particular, down to the present day, confound this with other renal maladies. Moreover, the latest English and French authors who recognize the individuality of this affection, differ among each other concerning the genetically different cases which are or are not to be included in it. For the present, I must give up all attempts to determine its frequency by statistics, since my own cases are too few in number to be of any independent value, and the data of other observers, for the reasons I have given, cannot, in my opinion, be made use of. In confirmation of this, I may refer, for example, to Lecorché's account of the etiology of his *néphrite parenchymateuse profonde ou grave*, on p. 166 of the above-mentioned work, where all the known causes of the Bright's disease of the old school, which he is seeking to set aside, are peacefully ranged side by side.

Young persons are decidedly more frequently attacked with this disease than those who are advanced in years. Among my own cases there was only one man who was over fifty years of age, and, on the other hand, many of the patients were children.

According to Dickinson, the affection is much more frequent in men than in women.

Summary of the Course of the Disease.

Of the cases of chronic nephritis, only a small minority are ushered in by an acute attack; and, so far as these are concerned, I have nothing to add to what has been already said concerning the initial symptoms of acute parenchymatous nephritis and the diseases which precede it.

When inflammation of the kidneys insidiously supervenes in one of the above described chronic processes, its commencement will only be disclosed, as Case XVI. shows, by systematic examinations of the urine, which it is a standing rule in our medical clinic to institute in every suspicious case of disease.

Nothing betrays the insidious affection of the kidneys in its earliest stage; there is no pain or discomfort of any sort in micturition, the only signs being a diminution in the quantity of urine passed daily, and the presence of albumen. The case detailed above shows how small the quantity of urine may be, and how large the percentage of albumen. Later on I will give more minute details concerning the characters of the urinary secretion in this affection, from the results of my own examinations. Here I will only remark that a diminution in quantity, and a large percentage of albumen in the urine, may be observed for a long time, and that the excretion will not revert to its normal conditions until the inflammation subsides. I will presently also explain why this does not occur in every case. In those cases in which, despite the existence of circumstances which might possibly lead to the development of this disease of the kidneys, no examination of the urine is instituted, and in those in which the disease attacks, without evident cause, persons who were previously in sound health, dropsy is, almost without exception, the first symptom that betrays the malady. It is true that when these patients, frightened by the commencing dropsy, seek medical assistance, we find them, as a rule, noticeably pale and anæmic, although they had previously considered themselves perfectly well, were not suffering from any disease of the bones or other chronic suppurative process, and had passed through no malarious disease. They usually state that they have noticed for some time past a diminution of bodily strength, which, however, has not been sufficient to prevent them from pursuing their occupations. The patients, as a rule, have experienced no other disturbances of health which would point to any decided organic disease. In a few rare cases they complain of dull, pressing pains in the renal region, which, however, masked under the customary cloak of *rheumatism*—that devil's dam in medical diagnosis, the familiar *bête noire* of the slothful and ignorant physician—fail to excite the suspicions of the patient, and too often also of his medical adviser.

Once begun, the dropsy is wont to increase rapidly, and usually, despite all therapeutic measures, attains an extreme grade. Its distribution is the same as that of all the other forms of renal hydrops, the subcutaneous areolar tissue being its chief seat. Beginning either in the feet or in the face, it extends, as a rule, over the entire body, and usually holds its ground obstinately in the parts it has once involved. The external genitals, in particular, are constantly swollen, and often remain swollen for months at a time, so that the foreskin is curled up in front of the penis like a post-horn, and the scrotum presents the appearance of a bladder filled with water, which may be larger than a child's head, and can then no longer be accommodated between the swollen thighs. Very generally the abdominal walls are involved to an extreme degree in the general swelling, and the abdomen appears to be enormously distended, even before any perceptible quantity of fluid is collected in the peritoneal cavity.

The serous cavities, however, do not by any means remain free from dropsical collections. Large quantities of fluid are more frequently found in the pleuræ, the pericardium, and the abdominal cavity, in the chronic than in the acute form of nephritis; and it is not at all rare for death to be caused by them, when the fluid is not evacuated at a sufficiently early period.

In no other disease of the kidneys have I so often observed extreme hydrops anasarca as in the chronic parenchymatous inflammation. It has been principally in cases of the latter affection that I have met with rupture of the epidermal layer in consequence of the enormous tension of the skin; in these cases I have seen the dropsical fluid trickle out from the small cracks in such quantities that the entire bed of the patient was wetted through and through, and the fluid which had filtered through the mattresses had collected in pools on the floor. The epidermis over an extensive surface is macerated in this fluid, and thrown off, leaving the corium bare and exposed; this is observed most frequently on the lower part of the thighs and in the scrotum. In the favorable cases a general diminution of the dropsy follows this enormous discharge of fluid. The bare and relaxed corium in the affected spots is covered with pale, glassy granulations, over which a new layer of epidermis is developed; the skin in these spots then looks as if it were covered with warty growths. In other cases, however, the spots in which the corium has been deprived of its epidermis become the starting-points of deep or superficial gangrene. In this way I have several times witnessed a gangrenous destruction of almost the entire scrotum; in all these cases, however, the testicles, which have been left entirely bare and unprotected, are very soon covered with granulations, and by the cicatricial contraction in every instance a cutaneous investment for these organs was reproduced. Another sequel of the loss of the epidermis, less common, however, than superficial gangrene of the skin, is a phlegmonous inflammation of the subcutaneous cellular tissue, which, as a rule, proves fatal.

The mucous membranes are also involved in the dropsical swelling, especially the membrane lining the intestinal tract. Vomiting of watery masses and profuse watery diarrhœa are the symptoms which indicate this condition of the gastric and intestinal mucous membrane. In consequence of this swelling, the epithelium of the intestinal mucous membrane may be destroyed, like the epidermis on the most swollen parts of the outer skin, and extensive ulcerations of the membrane may result therefrom.

The mucous membrane of the respiratory organs seems to be less frequently affected than that of the intestines. It may be mere chance, but I have as yet encountered not one single case of œdema of the glottis in connection with chronic parenchymatous nephritis. Œdema of large portions of the pulmonary tissue occurs more frequently, and, as a rule, proves fatal. Dropsy is very seldom entirely absent in chronic parenchymatous nephritis. According to Johnson's statements, it was only absent twice in one hundred cases of this disease. I have in my own clinical wards at the present time a butcher boy, seventeen years of age, who after suffering for a long time from ague was attacked with nephritis nine months ago, but who exhibits as yet no trace of dropsy.

The advance of the dropsy will not be checked until the excretion of urine again becomes abundant, consequently not until the inflammatory process in the kidneys begins to recede; not until then will we have reason to expect that the dropsy will gradually disappear. If it has once attained a high grade, a long time invariably elapses—sometimes more than a year—before it entirely and permanently disappears. Even then, however, if the urine continues to contain albumen, as is unfortunately often the case, there is always danger that an exacerbation of the renal disease may set in, in consequence of which the urinary secretion will once more be arrested, and the dropsy will return. As long as the dropsy exists, it conceals the extreme emaciation which has set in meanwhile, both from the patient and his physician. Sometimes it is astonishing how skeleton-like the previously shapeless and swollen limbs become when the dropsy has entirely disappeared; not only the subcutaneous adipose tissue, but also the muscles, have been reduced to the merest remnants.

The patients, who were previously unable to stir on account of the unwieldy swelling, find themselves excessively enfeebled when they attempt to use their limbs again after the dropsy has subsided. The faded, ashy color of the previously swollen face, and the paleness of the mucous membranes wherever visible, are evident symptoms of extreme anæmia. When complete recovery takes place, which is rarely the case, the patients slowly improve in condition, and it is a long time before they recover their former healthy aspect. The convalescence is more frequently incomplete. A portion of the renal tissue has been destroyed, and what is left continues to excrete albuminous urine. The patients then remain somewhat emaciated, the unhealthy color persists, the skin is constantly dry, and the hair thin and rough. They do not completely recover their strength, although some of them are able to return to their ordinary employments. Sooner or later death is caused by the consequences of this condition.

It is only in the very mildest cases, where there is no dropsy, that the impairment of the general nutrition is not very apparent As long as the patients remain free from secondary inflammations in other organs, the chronic parenchymatous nephritis runs its course entirely without fever. The behavior of the circulatory organs varies greatly, according to whether the disease attacks persons who are previously healthy and robust, or those who are already weakened and reduced by other diseases. In the former case, moreover, it is different at the commencement of the malady from what it is at a later period when a considerable degree of anæmia has been developed. In persons who were previously robust and vigorous, the pulse at the beginning of the renal inflammation is usually remarkably slow, full, and tense, and the heart-sounds loud and sharp. At a later period of the protracted disease, when the tension within the vessels has been lessened by extensive transudation of water, the pulse becomes weaker, less tense, and at the same time more frequent, and the cardiac sounds become less distinct.

In previously enfeebled individuals both pulse and heart-sounds have these characters from the very first. It must be remembered, however, that in many cases the enfeeblement of the heart-sounds is partly due to the resistance offered to the conduction of the sounds by the fluid that has accumulated in the pericardium and by the œdema of the thoracic walls.

Finally, even when the inflammatory process in the kidneys subsides, complete recovery does not take place in every instance, because oftentimes a large portion of the renal tissue has been destroyed in the course of the inflammation. In such cases the kidneys after death present the condition which I will describe presently, and which I think is most appropriately designated by the term *secondary atrophy*. These kidneys, after the inflammation has ceased, continue to excrete an albuminous urine, because a great part of the secreting vessels have been obliterated, and those which remain carry on their functions under an abnormally high blood-pressure. In such cases, while the general nutrition improves, a secondary hypertrophy of the left ventricle, with its train of consequences, is developed in consequence of the obliteration of so many peripheral arterial branches.

No disturbances of respiration are complained of by the patients so long as the functions of the respiratory apparatus are not interfered with directly by œdematous swelling of the duplicatures of the mucous membrane at the entrance into the larynx, or by accumulations of fluid in the pleural cavities or in the alveoli of the lungs, or indirectly by hydrops ascites. In those patients who are constantly confined to bed in consequence of the dropsy, no great demands are made upon the breathing capacity. Nevertheless, they also suffer from terrible dyspnœa as soon as the pleural cavities, the pericardium, or the abdominal cavity are filled with water in any considerable extent, or as soon as a large portion of the pulmonary tissue is inundated by œdema. Pulmonary œdema is one of the more frequent causes of death in chronic nephritis also. Besides the sense of oppression caused by increasing difficulty of respiration, the long agony is preceded by a tormenting cough, accompanied by profuse, watery, and frothy expectoration, and râles in the bronchi which can be heard at a distance; by a constantly increasing cyanosis of the lips, which contrasts strangely with the ash-pale color of the swollen face; by a disappearance of the pulse, and by a gradually ascending coldness of the extremities. The death-struggle is occasionally interrupted by repeatedly recurring epileptiform attacks, which occur even in cases in which the existence of increased tension in the aortic system had long been out of the question.

Digestive disturbances set in pretty early in some cases, and a failing or capricious appetite, or slight dyspeptic symptoms, may attract notice even before œdema directs attention to the kidneys. The patients, with especial frequency, experience a positive repugnance for animal food. While the anasarca is increasing, most patients complain of a want of appetite and eat therefore very little, but feel heavy and uncomfortable even after taking very moderate quantities of food. Others, on the contrary, continue to eat very good meals and appear to digest them well. I saw a boy of thirteen whose entire body was swollen to a shapeless mass, and who was unable to open his eyes on account of the enormous œdema of the eyelids, daily consume the full diet of our hospital with the greatest possible relish. When the dropsy is at its height, vomiting is a frequent symptom; it does not occur especially after eating or drinking, but oftener in the morning soon after awaking. The vomited matter consists of watery, slimy masses, which generally have a slightly acid reaction, and contain an exceedingly small quantity of solid constituents. In one such case I found the specific gravity of the vomited fluid to be 1002. I have already stated it as my opinion that this vomiting is the result and symptom of œdema of the mucous membrane of the stomach—an opinion which is based on anatomical evidence. The same may be said of the connection of the obstinate diarrhœa, which is observed in some of these cases, with the analogous state of the mucous membrane of the intestines. This diarrhœa is generally very profuse, and the stools, which are at times dark, and at others scarcely colored at all, occasionally contain large quantities of nearly unmixed pus and shreds of necrosed mucous membrane, and are then horribly fœtid. As above remarked, this peculiar character of the stools depends upon the so-called *Secondary Dysentery and Ulceration* of the Intestines.

In most of the patients vomiting and diarrhœa do not occur at all; in nearly all of them, however, the appetite is diminished, and is only restored when the other symptoms indicate a retrogression of the renal inflammation, *i. e.*, when the urine becomes more abundant, the albumen disappears, and the dropsy subsides. When the normal appetite returns, the general nutrition invariably improves, and the natural plumpness of the body, the fresh color of the skin and the mucous membranes, and the habitual degree of strength, are gradually restored.

Actual uræmic symptoms, epileptiform attacks, coma, amaurosis, etc., are observed much less frequently in chronic parenchymatous nephritis than in the acute form or in primary atrophy of the kidneys. In by far the larger proportion of cases these symptoms are entirely absent, although in some few cases I have seen them occur, with the most fearful intensity, just at the height of the malady, so that the shapelessly swollen body was shaken by the most violent convulsions. In one of these cases death ensued during coma, which had been preceded by a rapid succession of epileptiform attacks. In another case the uræmic symptoms did not make their appearance until the kidneys had passed into the stage of secondary atrophy, and they then proved fatal.

More common, however, than these uræmic nervous symptoms in chronic parenchymatous nephritis, are the *secondary inflammations* in other organs, which, like Traube, I attribute likewise to the contamination of the blood with urinary constituents. Indolent infiltrations of the pulmonary tissue, which do not follow the typical course of genuine pneumonia, and suppurative phlegmonous inflammations, are frequent causes of death.

Summary of the Post-mortem Appearances.

It has been repeatedly stated that the chronic parenchymatous nephritis in some cases follows the acute form of the disease. It follows from this that the pathological changes which the kidneys undergo in respect to their anatomical structure in the chronic cases, must be closely allied to the changes which we discover in the acute inflammations of the same organs, and, in fact, they only differ from the latter because the longer duration of the process allows the signs of inflammatory new formation on the one hand, and of inflammatory destruction on the other, to attain a higher grade.

The post-mortem appearances also vary, according as death sets in when the disease is at its height, or when the pathological process has passed through its entire course, or has, so to speak, exhausted itself. When the disease proves fatal, as usually happens, at its height, and before the above-mentioned termination is attained, we find both kidneys very considerably enlarged (provided one, or, as happened in one of my cases, a part of one, had not been previously atrophied), and, as a rule, much larger than they ever are in acute nephritis. "When only one kidney is present, or when the other has become incapable of performing its functions," says Klebs, "the diseased kidney, which may entirely fill the hypochondriac space, presents rather the appearance of a tumor ; at all events, the enlargement is more excessive than it ever is in cases of cyanotic induration and simple granular degeneration, and the organ not unfrequently attains double or treble its ordinary volume."

The capsule is tightly stretched, and gapes widely upon section. As a rule, the capsule, which is usually very delicate and thin, in consequence of the great distention, can be easily separated from the parenchyma of the organ. Here and there small particles of soft parenchyma sometimes remain attached to it. Hence, even after the capsule is stripped off, the surface of the kidney appears perfectly smooth, or only presents slight inequalities wherever little portions of the tissue have remained sticking to the capsule.

These large kidneys are always exceedingly anæmic on the surface ; their color, therefore, is always strikingly pale, almost white, with a strong tinge of yellow. In strong contrast to this yellowish white background, stand out the bluish red, stellate venous radicles, which are greatly distended with blood. The whole cortical substance exhibits on section the same anæmic pallor, the same yellowish white color as the surface, and contrasts sharply with the likewise enlarged, but often dark red pyramidal substance.

Klebs thus describes the appearance of the renal parenchyma as seen from the surface: "Upon close inspection we can distinguish numberless dull white granules, lying in a clear gray and often almost gelatinous ground substance. These granules are pretty equably distributed over the surface, but vary greatly in shape; sometimes they seem to be made up of smaller particles, sometimes we can recognize spiral lines which frequently connect the separate granules with each other ; they are the convoluted tubes of the cortical substance filled with fatty epithelium, these tubes in the outer layers of the cortex not presenting the regular arrangement which is found on vertical section of the organ.

"When a vertical section is examined, we see in the first place that the enlargement of the kidney is principally due to an increase of the cortical substance, which is double or even three times its ordinary thickness. The isolated elements which give to the surface of the organ the above described appearance are here more separated from each other, and we are able to make out better the condition of the isolated parts; dull white striæ arranged perpendicularly to the surface, alternating with somewhat broader gelatinous striæ; the former, the zones of the convoluted tubes, contain the glomeruli—which are at first tensely distended and later on become collapsed, and the small extravasations (?); the latter contain the less altered straight tubules, the swelling of the ground substance being only apparently more distinct than in the other zone. The dull white lines of the curling tubuli extend a short distance into the medullary substance, penetrating in between the bundles of the vasa recta and of the straight tubules at the bases of the pyramids. The latter are consequently considerably broader than usual, but otherwise present no notable changes."

Klebs likens the consistency of the organ to that of caoutchouc, which it is said to resemble in regard to its capacity of elastic resistance. To my mind this comparison is not suitable. I shall describe its consistency rather as doughy, like that of a fatty liver.

The mucous membrane of the pelvis of the kidney presents, as a rule, catarrhal swelling and a slight degree of hyperæmia.

The microscopical examination of the cortical substance of the kidneys, which macroscopically present the above described alterations, reveals the same changes which we have already met with in acute parenchymatous nephritis, only in a more marked degree. They involve both the tubuli uriniferi with their epithelium and the intertubular substance. The tubuli for the most part are considerably dilated, but a few are found here and there which retain their normal calibres. The epithelium of the tubes is only partially preserved, and the cells which still remain are greatly enlarged and are often rendered so opaque by the fat particles and minute oil-globules which they contain, that the nuclei can no longer be seen.

In many places the epithelial lining of the tubules is completely gone, and in its stead the tubules are entirely filled with masses of detritus mixed with oil-globules. Such a tube, when examined under the microscope by reflected light, sometimes appears perfectly dark and opaque, and is often bulged out unequally in different places.

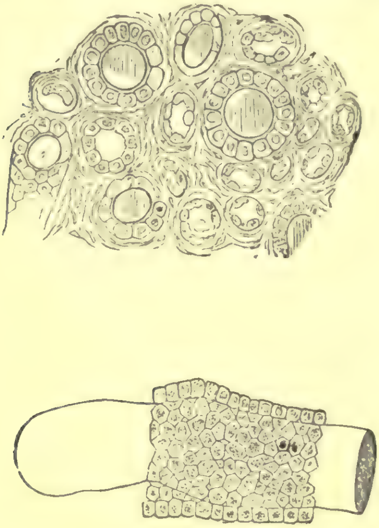


FIG. 8.

Waxy, highly refracting casts in the lumen of urinary tubules that still retain their epithelial lining. (Colberg.)

It is not uncommon to to find the lumina of some of the urine tubes blocked up with casts ; at the same time the epithelial lining of the tube at the affected part may be perfectly preserved and present its normal features, as Fig. 8 shows. In some cases we find an enormous number of the straight tubes filled with casts of this kind, when they usually have a slight yellowish color and a wax-like brilliancy.

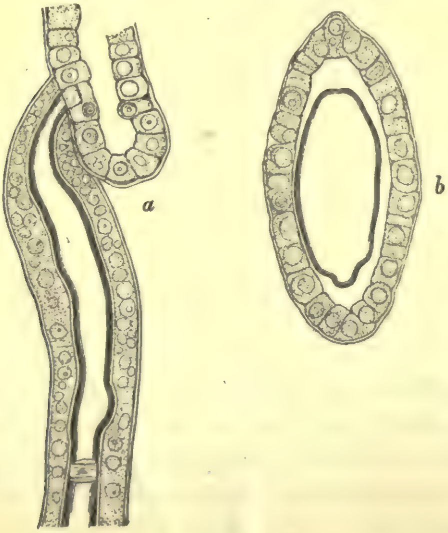


FIG. 9.

Waxy, highly refracting casts in the interior of straight tubules. Epithelial layer well preserved. (Heller.)

Fig. 8 is drawn from a preparation of my deceased colleague Colberg ; the preparation is from the kidney of a woman who died in my Clinic, of chronic parenchymatous nephritis. This, with some other drawings made use of in this work, were most kindly assigned to me by Colberg's bequest.

The interspaces between the tubuli are much broader than normal, according to Klebs, from two to four times as broad; the same author says that they often become as thick as the convoluted tubes themselves. The figures 9 and 10, the latter also after a drawing by Colberg, illustrate this condition.

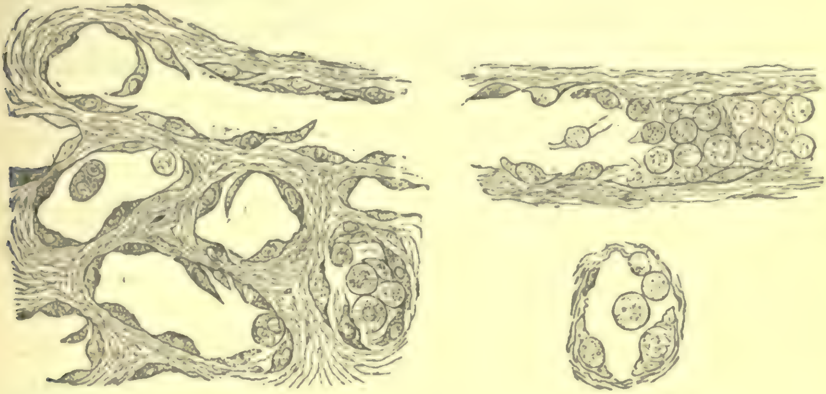


FIG. 10.

Section from the cortical substance. Enlargement of the intertubercular spaces; the walls of the tubuli are covered with a new-growth of epithelial cells. Chronic parenchymatous nephritis. (Colberg.)

This thickening of the intertubular matrix is partly due to its saturation with fluid exudation, and partly to the multiplication of pre-existing connective tissue elements under the stimulus of inflammation, and finally, in some part to the migration of numberless white blood-cells. The latter undergo in part a fatty degeneration, and of the cells which have undergone this metamorphosis there finally remains only a little mass of fat-globules. Sometimes a large number of these little masses are found in the intertubular tissue of the kidneys; their relative positions as regards each other betray their origin from migrated and degenerated lymph cells.

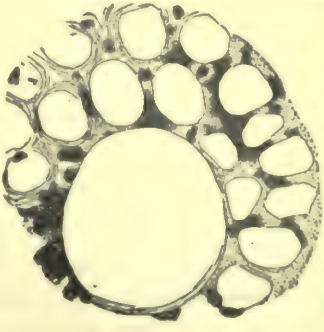


FIG. 11.

Collections of fat globules in the intertubular spaces. Section from a kidney in which parenchymatous nephritis was complicated with amyloid disease of the vessels. Magnified 150 diameters. (Colberg.)

We find, with remarkable frequency, the small arteries and Malpighian vascular tufts in chronically inflamed kidneys in a condition of amyloid degeneration, although, at least according to my own experience, only in the cases that succeed chronic suppurations or syphilis. I will presently explain the importance of this complication.

The bodies in which the large white kidneys which I have just described are found are always more or less dropsical. I have never met with such kidneys in a body that was not anasarcous.

The rest of the post-mortem appearances vary, in the individual cases, according to the exciting cause of the nephritis and according to the complications to which the renal affection has led: splenic tumors, diseases of the bones and joints, extensive cutaneous ulcers, abscesses in the cellular tissue, pulmonary consumption, purulent effusions into the serous cavities, pneumonia, etc.

Also in the chronic form of parenchymatous inflammation of the kidneys resolution may take place and the normal functions of the organs may then be once more established. This, however, only seems to be possible when the process has not lasted for a very long time, and when the determining cause that led to the renal disease can be removed and the other injurious influences be warded off. I have seen complete recovery take place in a case of nephritis following scarlet fever after the affection had lasted eighteen months; and again in a case of nephritis due to a severe cold, in which the patient had been bedridden for a whole year on account of extreme dropsy.

That the re-establishment of the normal functions of the kidneys in these cases depends upon the restoration of the normal structure of the organs appears to me a matter that is beyond all doubt.

Here and there some tubules or groups of tubules may be destroyed—perhaps all in which the degeneration of the epithelial linings has been complete; but this does not prevent the regeneration of the remaining parts. Provided the loss of secreting substance has not been too great, the functions of what remains suffer no disturbance. In my prefatory remarks I directed attention to the fact that nature in constructing the body of man has supplied with lavish hand the apparatus destined to excrete the urine, so that, even though one entire half of it be destroyed, the half that remains is capable of fulfilling perfectly the task of both. With regard to the way in which the restoration to the normal condition takes place after long-continued parenchymatous inflammation of the kidneys, we are as yet only able to offer conjectures. Probably, however, the process is analogous to that by which the resolution of an acute inflammatory process is affected, *i. e.*, that a new growth of epithelial cells from the walls of the urinary tubules replaces the diseased and destroyed cells. The investigations of L. Mayer and Kelsch prove that this takes place in the cholera kidney; Ottomar Bayer[1] also entirely confirms the statement made by Axel Key and supported by ample proof, that an active regeneration of the renal epithelium takes place; he further states "that very threatening defects may be repaired, principally by the proliferation of the cells which remain, although in many chronic cases the tunica propria and the intertubular tissue take part in the restorative process." This condition cannot, it seems to me, be more satisfactorily portrayed than it is in Fig. 8, after a drawing by Colberg; it shows the walls of the dilated urinary tubules clad with small and flattened epithelial cells.

The fluid that has infiltrated the intertubular spaces can be carried off by the lymphatics as soon as the pressure within the vessels subsides, and a part of the lymph-cells may also be removed by the same channels; some of these last may have already undergone degeneration, and their detritus may then be removed by absorption.

A perfect recovery, however, does not take place in every case when the inflammatory process subsides. Too much of the secreting substance of the kidneys has been destroyed; the long-continued inflammatory hyperplasia of the interstitial tissue has resulted in an increase of volume and in its organization into a firm, fibrous tissue, and in consequence of the pressure which the callous tissue thus developed exerts upon the blood-vessels, hitherto pervious glomeruli may be obliterated and a further secondary atrophy of the secreting parenchyma of the kidney may be the result.

---

[1] Archiv für Heilkunde. 1868. S. 148.

The chronic parenchymatous nephritis terminates in secondary atrophy of the kidneys, and the renal tissue that remains is no longer sufficient to carry on the normal functions of the organ.

With the subsidence of the inflammation in such cases, the dropsy, it is true, disappears; but it often returns at a later period, although the relapses are of slight intensity and transient duration. The patients, however, remain sickly, and die an early death, either from uræmia or from some secondary inflammatory affection.

The bodies of such persons are not usually dropsical, but the well-known cicatricial lines on the skin of the abdomen and thighs betray the previous existence of extreme dropsy. The kidneys are much smaller than when the malady is at its height, but nevertheless are rarely much smaller than normal kidneys; more frequently they still appear to be somewhat enlarged. Their consistence is uniformly firm and tough. The thickened capsule adheres firmly to the surface of the organ, and is not easily stripped off; small portions of the renal substance usually remain attached to it. The surface of the kidney is uneven and nodular, from contraction of the connective-tissue islands of the renal tissue, which vary greatly in size, projecting between the depressions. These prominent parts are often pale, and retain the yellowish tinge which the parenchyma presents at the height of the complaint, while the bands of connective tissue between them have a more whitish color. Still, I have seen the entire kidney, in cases of this kind, present a strikingly dark-brown color.

In the urinary tubules which are still in a good state of preservation, the microscopical examination shows the epithelium to be perfectly normal, and the same is to be said of the Malpighian tufts attached to them.

Besides these normal remnants of the secreting tissue, however, we find dilated and irregularly bulged-out tubules, in parts presenting the aspect of tiny cysts of about the size of a pin's head, or more, filled with clear contents or with a fatty detritus. Near these we find the remains of destroyed glomeruli, which appear as dark, roundish bodies, surrounded by concentric layers of spindle-cells. The intertubular spaces are considerably wider than normal, and we also find bands of perfectly organized fibrous connective tissue, where the true glandular substance has been entirely destroyed. The condition of the general nutrition of the body may be tolerably good, if the patient improved to a certain extent after the cessation of the actual inflammatory process, and no trace of dropsy, as already noticed, may be perceptible. In all these cases, however, the left ventricle is hypertrophied. If death has not been caused by uræmia, the autopsy shows pneumonia, or pericarditis, or pleurisy to have been its immediate cause. I have never known death to occur in these cases from apoplectic effusion.

Although nearly all the recent writers who have published accounts of the pathological histology of the large white kidney (of chronic parenchymatous nephritis, according to my nomenclature) agree in all essential points in their descriptions, if we except Kelsch, there is still very little harmony in the interpretation of the facts that have been observed. Especially discordant are the views concerning the order in which the changes in question are developed, and concerning the nature of certain of the processes which lead to those changes. Since the appearance of Arnold Beer's pamphlet "On the Connective Substance of the Human Kidney in its Normal and Diseased Conditions," Berlin, 1859, most of the German pathologists have, in my opinion, attached too exclusive importance to the changes in this connective tissue, and have in some measure habituated themselves to look upon the alterations of the renal epithelium as a secondary process, as a simple fatty degeneration evoked by defective nutrition.

Hence it is that the name "*Interstitial Nephritis*" is frequently given by preference to this process; the traditional belief in the three stages of Bright's disease may have had some influence in determining the acceptance of this designation.

I cannot agree with this view myself, and I base my objections to it especially on the fact that in acute parenchymatous nephritis, which, as we know from experience, occasionally passes into the chronic form, we find at the post-mortem exactly the same lesions in the kidneys as in the cases which are chronic from the very commencement.

In the milder grades of acute parenchymatous nephritis, such as we not unfrequently meet with at the post-mortem table after scarlet fever, death having resulted not from the renal disease, but from some complication, we sometimes find the epithelial cells already distinctly clouded and swollen by a granular deposit, before any noteworthy alterations have taken place in the intertubular connective tissue. This is a fact which admits of no contention, according to my own observations, and it seems to me to prove satisfactorily that the epithelium takes part from the very beginning in the inflammatory process, by an absorption of exudation into the cell-bodies, and that the term *parenchymatous inflammation* applies with equal correctness to the analogous changes in cases that pursue a chronic course. At the same time I do not wish to deny that the great tension of the fibrous capsule of the kidney, caused by the swelling of the whole cortical substance, produces ischæmia of the cortex and collateral fluxion to the vessels of the medullary substance, and thereby promotes the fatty degeneration of the epithelial cells that have undergone inflammatory swelling. This, however, does not invalidate my theory that the earliest changes occur in the epithelial cells.

The views of the most recent French writers are directly opposed to the ideas and statements of German authors upon this matter. Lecorché describes his *néphrite parenchymateuse profonde ou grave* as an inflammatory process which involves principally the epithelium—a true parenchymatous inflammation in Virchow's sense, the interstitial tissue being but little or not at all affected. He attacks Frerichs on account of his assertion that the hyperplasia of the connective tissue is not without influence on the altered condition of the kidney in this disease.[1]

---

[1] *Lecorché* quotes me in support of his assertion that the interstitial tissue remains unaltered in parenchymatous nephritis. I do not know how this misunderstanding arose. In No. 25 der Sammlung klin. Vorträge, S. 33, I have stated exactly the reverse.

Kelsch also denies that any change takes place in the interstitial tissue in the large white kidney. I am unable to account for these conflicting statements concerning a plain matter of fact.

Kelsch, however, as I have already stated, even disputes the inflammatory character of the changes he observed in the epithelial cells of such kidneys, and prefers to regard them as the expression of an anæmic necrosis, as the result of general malnutrition. Unfortunately, here Kelsch omits to explain why this ischæmic degeneration of the renal epithelium should be constantly absent in exactly those diseases in which the general marasmus is most intense—for instance, in slowly progressive occlusion of the orifices of the stomach and œsophagus, and in cancerous affections of other organs also.

Analysis of the Symptoms.

An exhaustive examination of the symptoms necessitates of course an investigation of the disturbances in the functions of the diseased organs, since they are the source of all the remaining symptoms which are observed in the course of this disease of the kidneys. It is necessary, I think, to describe with special minuteness the changes which the urinary secretion undergoes in this complaint, because, as it seems to me, neither in practice nor in the literature of the subject has the consideration hitherto been awarded to them which their importance with regard to the pathology, and more particularly with regard to the diagnosis, demands.

As long as the chronic inflammation of the kidneys continues to advance, or remains at its height, the quantity of urine excreted daily is far below the normal.

The complete anuria which is met with in some cases of acute nephritis I have, however, never observed in the chronic form. The diminution of the quantity of urine in the latter may persist for a very long time. In one of my patients the average quantity passed daily during the first nine months he was under treatment, as estimated from one hundred and twenty-two careful examinations, was only 698 c.c. Although, however, I may at once lay it down as an absolute rule that the excretion of urine at the height of the chronic nephritis is reduced to a very abnormal extent, the quantity passed in twenty-four hours may vary greatly within a very short period, and even on successive days; so much so that the quantity may be extremely small on one day and abundant on the next.

As soon, however, as the chronic inflammation of the kidneys is in process of retrogression, the quantity of urine excreted daily increases, and in the later period of the complaint and during the stage of secondary atrophy which sometimes follows, it may exceed the normal average.

I have at my command a large number of urine tables prepared in my wards, and having reference to cases of chronic parenchymatous nephritis, which illustrate this fact clearly. I will give here a few abstracts from these tables, with short notices of the course followed by the disease.

K., a day-laborer, from Denmark, thirty-three years of age, had suffered repeatedly from ague during several years, at one time for half a year without intermission. From June, 1867, he was dropsical; admitted into the hospital for albuminuria and general dropsy towards the end of November in the same year. Diagnosis: chronic parenchymatous nephritis (confirmed subsequently by the autopsy). Death on January 7, 1868, from pneumonia and œdema glottidis. During the six weeks he was in the hospital the quantity of urine he passed could only be estimated on twenty-four days, because on the other days some of the urine was lost through the patient's own neglect. Smallest quantity passed, 400 c.c.; largest, 1,000 c.c.; average estimated from these twenty-four days, 813 c.c.

Mrs. P., aged twenty-five years, wife of an inn-keeper on the moors, had suffered from intermittent fever during the entire summer of 1867; in the spring of 1868 she became dropsical. In August of the same year she came to Kiel, being then excessively dropsical, and was taken to a private infirmary. The enormous amount of albumen contained in the urine left no doubt of the existence of a parenchymatous nephritis. Between the middle of August and the 20th of September, the entire quantity of urine passed in the twenty-four hours was by good fortune collected upon twelve days. The smallest quantity passed was 270 c.c., the largest 500 c.c.; the average of the twelve observations was 400 c.c. per diem.

The dropsy was quickly and entirely subdued by hot-air baths. The patient, who was self-willed and homesick, would not stay any longer at the hospital, and left about the middle of October, although her urine still contained one per cent. of albumen. During the latter half of September I was absent for fourteen days, and the urinary analyses were discontinued during that time. After my return in the beginning of October I succeeded in collecting all the urine passed on seven days.

During this time the smallest diurnal quantity was 1,280 c.c., the largest 1,850 c.c., and the average of the seven analyses was 1,490 c.c.

The case already referred to in which the diminution in the excretion of urine persisted for a very long period (during nine months the average quantity, as estimated from 122 measurements, being only 698 c.c.), will presently be reported in detail, as it presents points of especial interest. In this case the renal inflammation terminated in secondary atrophy; the death was caused by uræmia after the case had been two and a half years under observation. During the last three months of life the urine was collected on twenty-five days; minimum, 700 c.c., maximum, 2,000 c.c.; average 1,085 c.c.

St., a boy thirteen years of age, admitted for general dropsy on the 5th of June, 1873. The œdema had commenced five days before his admission without any disturbance of the general health, and was, so far as was known, entirely unprovoked. Up to his death, which occurred on August 25th, during uræmic convulsions, the lad passed on an average, estimated from sixty-five measurements, 300 c.c. of urine daily, which to the very last contained a varying, but usually enormous quantity of albumen.

At the commencement of chronic parenchymatous nephritis, and while the malady is at its height, the color of the urine is ordinarily dirty brown, *and is darker in proportion to the smallness of the quantity.*

When the excretion is more abundant, the color may be quite light even while the disease is at its worst, but it always has a peculiar dirty tint. A bloody coloration is always exceptionally met with in a few cases, and is then merely transitory.

The scantier the excretion the more clouded it is, and the cloudiness may be perceptible as soon as the urine is passed and before it has had time to cool. It is due to the suspension in the fluid of often large quantities of formed elements (epithelium, débris, casts, white blood-cells), of which I will speak presently in describing the sediment.

A further clouding of the urine, which is secreted in scanty quantities at the height of the disease, almost invariably takes place as soon as it cools. This subsequent cloudiness depends upon the deposit of urates which such urine usually contains in great abundance. When the urine is heavy and viscid from the large amount of albumen it contains, the urates, though no longer held in solution after the fluid cools, instead of falling to the bottom of the vessel, remain suspended in the fluid, and make it as thick as muddy water. The uric acid crystals, however, which are usually formed in large quantities, fall to the bottom, or adhere everywhere to the sides of the urine vessel, which may thus be covered with a crystalline crust.

In the further progress of the disease the urine becomes clearer, and the relative quantity of uric acid and its salts contained in it becomes smaller in proportion to the increase in the quantity of the excretion. Still, the urine always deposits a more or less abundant sediment, the copiousness of which in the stage of secondary atrophy has sometimes astonished me. In such cases the bottom of the vessel that holds the urine is often covered with a layer of gray, powdery sediment that is several millimetres in thickness.

The microscopic examination of the sediment shows that, apart from uric acid and its salts and other crystalline structures, the principal ingredient is urinary casts, the quantity of which in many cases is really quite extraordinary.

Every drop of the sediment may contain them in dozens. I think I may safely affirm that the quantity of these casts increases with the duration of the disease. In examinations undertaken when the first traces of commencing dropsy appeared, I have sometimes found a few scattered casts after prolonged search; but, on the other hand, I have never seen them more abundant than in the sediment of the urine passed by a certain Mr. Kr. in the last few weeks of his life, when his kidneys were in the stage of secondary atrophy.

So long as the casts are scanty the greater number of them present characters which, in my opinion, prove that the malady is of recent existence: they are pale, hyaline, or slightly streaked, or dotted with isolated dark molecules or shining fat drops. We find thin, long, and slightly curved as well as broad casts, and to both sorts fragments of cells or white blood-corpuscles adhere. The longer the process has lasted, the more numerous become the dark granular casts, the greater the preponderance of the broad over the narrow casts, and the more abundant those peculiar broad yellow casts of wax-like refracting powers which, *e.g.*, I found in unusual quantities in the urine of the above-mentioned Mr. Kr. during the last weeks of his life.

Besides the casts, the sediment always contains white blood-corpuscles, often in very considerable quantities. Red blood-corpuscles, on the other hand, I have rarely found in the cases of chronic nephritis that commenced slowly and insidiously; even then they were only transient constituents of the sediment, although sometimes present in large quantities. In the cases which *commenced acutely* (for instance, after exposure to cold), the blood usually disappears after a few weeks; it may, however, continue present for months; but then, so far as my own experience goes, it seldom reappears in the subsequent stages of the disease.

Finally, we find in the sediment *flocculent masses of granular detritus*, that become more and more abundant as the urine diminishes in quantity. That these masses consist of an albuminous substance is evident from the fact that they, like the hyaline casts, assume a bright yellow color upon the addition of a watery solution of iodine and iodide of potassium. It is probable that they are the débris of the destroyed epithelial cells of the tubuli uriniferi, since after death we find many of these tubes completely filled with similar masses.

The specific gravity of the urine excreted in chronic nephritis varies with its quantity, rising and falling pretty regularly in an inverse proportion to the daily quantum.

At the height of the malady, when the smallest quantity is secreted, the specific gravity is regularly far above the normal. I have repeatedly found the specific gravity in such urine, as determined both by the pyknometer and the urinometer, to be above 1040—*higher consequently than the specific gravity of the blood-serum from which it was secreted.* This assertion is based not upon a mere assumption that the blood-serum of the dropsical patients from whom the urine was taken must necessarily have been abnormally watery, but upon the results of repeated examinations of the blood-serum of the patients in question. This serum was tested by the pyknometer, and its specific gravity found to be lower than that of the urine excreted at the same time.

For example, the boy St. was bled after the first epileptiform attack. The specific gravity of the serum I found to be=1018, while that of the urine passed shortly before was 1040.

The serum of the blood taken after death from the body of the nurse S. (Case XVI.) had a specific gravity of 1015.58; the specific gravity of the urine passed last before death was 1020.

Moreover, I obtained similar results when I was comparing the specific gravities of the blood-serum and of the urine in diabetic subjects.

As soon, however, as a more abundant excretion of urine sets in in chronic nephritis, the specific gravity falls, and it is often found to be abnormally low, even before the diurnal quantity of urine passed has reached the normal point.

The quantity of urine passed by Frau P. on four consecutive days in August, 1868, was respectively 375, 395, 300, and 325 c.c., with a sp. gr. of 1040, 1041, 1038, and 1039. On the 21st of September 500 c.c. were passed, which had a sp. gr. of 1020. In the *second week of October* she passed *daily, on an average*, 1,490 c.c., the sp. gr. on the different days in this week fluctuating between 1008 and 1012.

Herr Kr., whose long illness afforded me an opportunity of making continuous observations for a long time, passed in August, 1867, on *four consecutive days*, 490, 490, 400, and 390 c.c. of urine, with a sp. gr. of 1038, 1035, 1038, 1035. On the 5th of September in the same year he passed 1,066 c.c. of urine, with a sp. gr. of 1010. On Oct. 14th, 500 c.c.; sp. gr., 1030. On Oct. 15th, 350 c.c.; sp. gr., 1036. On Oct. 16th, 730 c.c.; sp. gr., 1019.

In the autumn of 1869, the dropsy having long since subsided and the renal disease having entered on the stage of secondary atrophy, this patient excreted on twenty-five days on which observations were made, on an average, 1,083 c.c. of urine daily (maximum, 2,000; minimum, 700). The sp. gr. on different days during this period varied between 1008 and 1012.

I will state here at once my opinion as to the causes of this low specific gravity of the urine under different circumstances. The urine, which at the height of his malady was secreted in normal quantities, was derived, as the dropsy proved, from an hydræmic blood; that, on the contrary, which was passed during the last stage of the complaint was secreted under an abnormally increased blood-pressure, due to the hypertrophy of the left ventricle that had developed in the interval; consequently, it was secreted with abnormal rapidity, and hence of necessity possessed a low specific gravity.

The chemical analysis of the urine in chronic nephritis invariably reveals the presence of albumen; this is never absent at any period, and when the disease is at its height the *percentage of albumen is greater than it ever is under any other circumstances.*

Cases occur in which, during the period of increasing dropsy, a quantitative estimate of the albumen contained in the urine by the ordinary method of boiling cannot be made, unless the excessively scanty urine be previously diluted with four or five times its volume of water.

If such urine be heated without previous dilution, the entire quantity exposed to the test coagulates into a stiff jelly long before the boiling point is reached. This coagulation makes a filtration impossible. In such urine *the quantity of albumen may reach to five per cent., and even more.*

As an example, I may refer once more to the above detailed case of the nurse L. S. (Case XVI.). This patient, on the 8th of November, 1873, passed only 135 c.c. of urine in the twenty-four hours. Its reaction was highly acid, and its specific gravity 1040. A measured quantity of this urine was *diluted with five times its volume of water, and heated to the boiling point.* At this temperature a moderate coagulum of albumen separated itself, which was collected upon a filter previously dried and weighed. It was then carefully washed, and dried, together with the filter, at a temperature of 100° C. It was finally weighed and the calculation made, which showed that the percentage of albumen in the urine examined was 4.9. Another portion of the same urine (30 c.c.) was filtered, and mixed with *five times its volume of alcohol*, which threw down a moderate coagulum. At the expiration of twenty-four hours the coagulum was collected on a previously weighed filter, and washed with alcohol until no traces of chlorides could be discovered in the filtered fluid, and was then dried and weighed. It was found that almost exactly two grammes of albumen had been collected upon the filter, which corresponded to *over six per cent. of albumen in the urine.* I must call attention here, however, to the fact that, when albuminous urine is treated with alcohol, the coagulum is always greater than when it is simply heated. In chronic nephritis, especially, the urine appears to contain an abundance of albuminates and other substances (inorganic salts, uric acid), which are thrown down by alcohol, but not by boiling.

The percentage of albumen in the urine in chronic nephritis is exceedingly variable, not only in different cases, but at different times in the same case.

It not only fluctuates with the advance or retrogression of the inflammatory process, but also, like the quantity and the specific gravity, is often different on successive days.

Its proportion to the specific gravity is a pretty constant *one, and hence, like the latter, it stands in an inverse ratio to the quantity of urine passed daily, rising or falling as this diminishes or increases.*

Frau P., in August, 1868, passed, upon four consecutive days, 375, 395, 300, and 325 c.c. of urine, the sp. gr. being 1040, 1041, 1038, and 1039 respectively, and percentage of albumen 4.12, 3.97, 4.18, and 4.06. Two months later the same patient passed, on an average, 1,490 c.c. of urine daily for seven days, the specific gravity varying on the different days between 1008 and 1012, and the percentage of albumen between 1.06 and 1.391. Nevertheless, *the absolute quantity of albumen lost each day during this latter period, when the excretion of urine was more abundant, was greater than in the earlier stage, when the excretion was scanty.* Frau P. lost, with the small quantities of urine passed on those four days, 15.45, 15.68, 12.54, and 13.20 grms. of albumen; two months later, when she was passing an abundant quantity of light urine, she lost, on an average, 17.90 grms. of albumen daily.

The day-laborer K., on December 1, 1867, when he passed 1,250 c.c. of urine, the sp. gr. being 1022, and the percentage of albumen 3.7, lost the enormous quantity of 46.25 grms. The urine passed by him on the day before his death from pneumonia was more highly albuminous than at any other time during his illness; there was 5.7 per cent., or 30.02 grms. of albumen, in 525 c.c. of urine, with a sp. gr. of 1029.

From a comparison of the urinary analyses of different cases, it is evident that the specific gravity of an albuminous urine does not, by any means, correspond with a certain fixed percentage of albumen.

The heaviest urine passed by Frau P. (sp. gr. = 1041), contained only 3.7 per cent. of albumen, whereas K.'s urine, which contained 5.7 per cent of albumen, had a sp. gr. of only 1029.

Even in the same case we are only justified in assuming that the percentage of albumen has increased or diminished, because the specific gravity has risen or fallen, when the changes in the latter take place within a very short period.

The nurse S., who on the 8th of November passed 135 c.c. of urine, with a sp. gr. of 1040 and 4.9 per cent. of albumen, passed on the 28th of the same month 155 c.c. of the same specific gravity, but with only 2.564 per cent. of albumen.

The following figures, taken from the urinary analyses made in my own wards, show *what large quantities of albumen are carried off by the urine in chronic nephritis.*

The day-laborer K., according to the results furnished by seventeen careful analyses of the urine made during the last month of life, lost on an average 17.26 grms. of albumen daily. Frau P., according to the results of eighteen analyses made within a period of two months, lost on an average 15.28 grms. daily; Herr Kr., according to the results of fifty analyses made during the first six months he was under our observation, lost 10.04 grms. daily. The boy St., according to the results of twenty analyses made within a period of two and a half months, lost 7.23 grms. daily.

As the disease progresses towards the stage of secondary atrophy there is a diminution not only in the percentage, but also in the gross quantity of the albumen contained in the urine.

In Herr Kr.'s urine, the specific gravity of which fluctuated, during the first six weeks he was under observation, between 1012.5 and 1035, the percentage of albumen was found to vary between 1.3 and 3 per cent. (the extremes corresponding with the extremes of the specific gravity), and the diurnal loss of albumen between 10.41 and 20.46 grms. Subsequently the percentage of albumen never rose as high as 3 per cent. The gross loss of albumen, however, on many days during the ensuing fifteen months, surpassed the average daily loss during the first six weeks, sometimes by as much as 4 or 5 grms. *The average daily loss of albumen in the period included between the* 1*st of January*, 1868, *and the* 28*th of May*, 1869, *as estimated from one hundred and ninety-two analyses, was* 84 *grms.* After April, 1869, at which date the patient had already been twenty-two months under observation, the *analyses never revealed quite* 1 *per cent. of albumen, and the diurnal loss scarcely amounted to half the average daily excretion of albumen in the beginning of the malady.* From the end of May to the commencement of September, 1869, the analyses were discontinued, but between the 4th of September, and the 12th of October, twenty thorough analyses of the urine passed in each twenty-four hours were made. During this period the percentage of albumen varied between 0.153 and 0.399 per cent., and the average entire loss on each day of observation was 3.3 grms. I have taken the trouble to add up the daily losses of albumen in Herr Kr.'s case, as reckoned from the two hundred and sixty-two complete analyses of the urine, which were distributed over a period of more than twenty-seven months. This addition gives the round sum 2,200 grms. of albumen for these two hundred and sixty-two days, an average loss per diem of 8.4 grms., and for the entire period of twenty-seven months, reckoning thirty days to the month, a total loss of 6,804 grms. (circa 18 lbs. Troy weight).

The quantity of urea contained in the urine in chronic nephritis, like the albumen, not only varies exceedingly both *relatively and absolutely in different cases, but also fluctuates very considerably in the same case.* The sum total of the excretion of urea depends in the first place upon the quantity of this substance produced in the system, and this in its turn, as is well known, depends upon the rapidity with which the tissue-changes take place during the period of observation. Since, however, this measure may be very different in different patients and in the same patient at different times, it follows necessarily that on this account alone the absolute quantity of urea carried off daily by the urine must be variable, entirely independently of any possible insufficiency of the kidneys, in consequence of changes produced by the disease in question, to perform their natural task of depurating the blood from this refuse product of the tissue metamorphosis.

With regard to *the percentage of urea* in the urine excreted by kidneys which are the seat of chronic inflammation, the analyses instituted by us have, however, demonstrated it to be *a pretty constant rule*, that the relative quantity of *urea to a certain extent rises and falls with the rise and fall of the specific gravity of the urine;* that consequently in the urine excreted by these diseased kidneys, just as in that excreted under other circumstances, the percentage of urea increases in an inverse ratio to the diminution in the daily quantity of urine passed.

In Mrs. P.'s case, for example, the heaviest urine, which had a sp. gr. of 1040 and 1041, contained the largest percentage of urea, namely, 4.8 and 4.9 per cent., whereas the urine passed two months later, which had a sp. gr. of 1008 and 1009 contained only the small percentage of 0.8 to 0.9 per cent.

In this case the maximum daily excretion of urea—18.96 to 18.38—coincided with the highest percentages, while, on the other hand, the minimum daily excretion did not coincide with the smallest percentages. Nevertheless, the average quantity of urea excreted per diem, during the first month the patient was under observation, when the urine was scanty and very heavy, was considerably greater than at a later period, when the patient passed much larger quantities of urine of a lower specific gravity. In the second half of the month of August the average quantity of urine passed daily, as estimated from urine analyses, was 380 c.c., the specific gravity of which fluctuated between 1032 and 1041, and the percentage of urea between 2.8 and 4.9 per cent., 14.2 grammes of urea being excreted daily on an average. In the second week of October, on the other hand, when urine was collected and analyzed every day, the average quantity passed was 1,490 c.c. (specific gravity, 1008–1012, and percentage, 0.8–1); but the average excretion of urea per diem was only 18.23 grammes.

In the case also of the day-laborer, K., the highest and lowest specific gravities (1035 and 1015) coincided with the highest and lowest percentages of urea (3.9 and 1.8 per cent.), and at the same time with the maximum and minimum quantities of urine (400 and 1,500 c.c.). The total quantity of urea excreted in the twenty-four hours, however, in this case, was less (15.6 grammes) on the day the smallest quantity of urine, with the highest specific gravity and the highest percentage of urea (400 c.c., with 3.9 per cent.), was passed, than on any other day during the time the patient was under observation; the largest quantity of urea excreted in any twenty-four hours was 28.75 grammes, and on that day 1,250 c.c. of urine, with a specific gravity of 1022, and 2.3 per cent. of urea, were passed. On this same day the total quantity of albumen excreted was larger than on any other day within a period of a month. According to an average estimated from sixteen analyses, K. excreted during this period 20.43 grammes of urea per diem in the urine.

In the case of Mr. Kr., too, the dependence of the percentage of urea upon the specific gravity of the urine was strikingly evident throughout the entire period that his case was under observation, although the highest percentage of urea did not coincide exactly with the highest specific gravity. In this case 4.0 and 4.1 per cent. of urea were found in urine with specific gravities of 1035 and 1038, while on one day within the first six months of observation, when the specific gravity of the urine was 1012.5, the percentage of urea was only 1.7. At a later period, when Mr. Kr. was passing larger quantities of urine, whose specific gravity never rose above 1012, the percentage of urea was never above 1.4 per cent., and it sank once, when the specific gravity was 1008, to 0.9 per cent. Simultaneously with this lasting reduction in the percentage of urea, the absolute quantity of urea excreted was permanently diminished.

A closer examination of the results obtained by the analyses of the urine made in the course of twenty-seven months, in the case of Mr. Kr., shows, however, that the quantity of urea secreted per diem was not solely dependent either upon the specific gravity of the urine, or upon the quantity of the excretion passed daily. The relation especially of the daily quantity of urea excreted to the daily quantity of urine is obviously altered as the renal disease progresses towards secondary atrophy. For instance, at the commencement of our observations in this case, the average daily quantity of urea increased as the quantity of urine increased, but it subsequently fell very considerably below its previous figure, in spite of a still further increase of the urinary secretion.

In order to prove this circumstance by figures, I have estimated and compared the average daily quantities of urine and of urea excreted during the different periods of the observation. *The first period* comprises the interval between the beginning of July and the end of December, 1867. During this period the urine was collected and measured on ninety-four days, and these measurements show 600 c.c. to have been the average quantity per diem. On fifty of these days the percentage of urea was determined, and the average quantity excreted daily was found to have been 16.5 grammes.

The second period takes in the time between the 1st of January, 1868, and the 28th of May, 1869, on which day Mr. Kr. left the hospital, the dropsy having meanwhile subsided, and went to the country. During this period the urine passed in twenty-four hours was collected and measured on two hundred and fifteen days, and the total quantity of urea ascertained on one hundred and ninety days.

From these measurements and analyses it was found that the average quantity of urine passed daily was 970 c.c., and the average quantity of urea per diem 20.60 grammes.

In September, 1869, Mr. Kr. returned to the hospital. Since he had left us at the end of May he had become very thin and pale, but the dropsy had not returned. Between September 4th and October 17th the urine was collected and measured, and the quantity of urea ascertained on twenty-five days. The result arrived at was that the average quantity of urine passed per diem was 1,018 c.c., and the average quantity of urea excreted daily was 11.75 grammes. That this quantity of urea did not correspond with the quantities in which this product of tissue metamorphosis was being formed in the system, notwithstanding the emaciation and the small amount of food taken, was evident from the fact that Mr. Kr. suffered during the whole of this time from symptoms of chronic uræmia, from uncontrollable vomiting and convulsive twitchings of the muscles. I was, however, never able to demonstrate the presence of urea in the vomited matter, the reaction of which was always acid.

I have not myself instituted any careful examinations to determine the relative quantities in which the other normal constituents of the urine are excreted in chronic nephritis. That the daily excretion of uric acid is often very considerable a mere glance suffices to show, since very generally the entire quantity of the urine after it has cooled is rendered cloudy by the urates, and the walls of the vessel in which the urine is collected are found covered with a crust of crystallized uric acid. The chlorides are at all events scanty in the urine of chronic nephritis.

The day-laborer K. excreted through the kidneys, on an average estimated from fourteen analyses, only 3.58 grammes of the chlorides daily. The urine of Mr. Kr. during the first period of the observation, contained a daily average of 3.557 grammes of the chlorides as estimated from thirty-one analyses, and during the second period 4.65 grammes as estimated from fourteen analyses.

I have made no examinations to determine the quantity of phosphoric acid excreted in chronic nephritis.

If now we group together the other symptoms that manifest themselves in the course of chronic inflammation of the kidneys —anæmia, dropsy, emaciation, and the ultimate termination in inflammatory processes in different organs or tissues, or in uræmic manifestations—and compare them with the disturbances in the functions of the kidneys caused by the disease, it will not be difficult to demonstrate the dependence of these symptoms upon these disturbances of function.

To commence with the most striking of the symptoms, the dropsy, my own investigations have convinced me that its appearance and subsidence exactly coincide with the diminution and increase of the daily excretion of water through the kidneys.

In each individual case where I was able to watch the commencement of the dropsy, it was preceded by a very abnormally diminished excretion of urine ; in each individual case, moreover, the quantity of water excreted through the kidneys continued to be below the normal average of health, or at least less than what would correspond to the water ingested in the food and drink as long as the dropsy was on the increase.

In the above detailed case of the hospital nurse S., who, even before the renal affection set in, was reduced to an extreme degree by inveterate syphilis and pulmonary consumption, on an average only about 700 c.c. of urine were passed per diem during the week before the first traces of œdema of the ankles were discovered.

Mrs. P. excreted, on an average estimated from twelve measurements, only 400 c.c. of urine per diem during the two months that the dropsical swelling was greatest. The day-laborer K., who was excessively anasarcous, according to an estimate deduced from twenty-four measurements, made during a period of six weeks, passed the average quantity of 813 c.c. of urine daily. Mr. Kr., upon an average estimated from ninety-four measurements made during a period of six months while the dropsy was on the increase, passed only 600 c.c. per diem. The boy St., who was exceedingly dropsical up to the date of his death, passed during the last two and a half months of life, on an average 300 c.c. of urine daily.

In the case of Mrs. P. the anasarca diminished considerably during the last few weeks of her stay in the hospital, and during this period she passed on an average 1,490 c.c. of urine daily. After Mr. Kr.'s return to the hospital, and during the three months which preceded his death, he was not at all anasarcous, and passed on an average 1,085 c.c. of urine daily; this figure, it is true, was calculated from the results of only twenty-five accurate measurements of the gross daily quantities.

I do not by any means wish to deny that other factors exert an influence on the development and disappearance of the dropsy; that, for instance, the impoverishment of the blood caused by the draining away of its albumen promotes the increase of the dropsy, while the intercurrent diarrhœas and the artificially provoked diaphoresis (diaphoresis does not occur spontaneously, on account of the ischæmia of the skin caused by the anasarca) favor its disappearance. This much, moreover, is rendered certain by Herr Rehder's observations and experiments: that the dropsy is due to the inability of the kidneys to remove the excessive quantity of water from the vascular system, and that it will not absolutely and permanently disappear until the renal functions are in this respect completely re-established, so that the quantity of water excreted through the kidneys will stand in the proper ratio to the quantity ingested in the food and drink. When the anasarca begins to subside, the quantity of water excreted by the kidneys is sometimes considerably greater than that which has been ingested, as Herr Rehder's tables show. The anæmia, and the consequent general loss of flesh and strength which are observed in patients suffering from chronic nephritis, are due to two different causes: the loss of the albumen which drains off in the urine, and the disordered appetite and digestion which are so constantly present. Between these two causes I do not hesitate a moment to give the precedence to the loss of the serum-albumen through the kidneys, notwithstanding the strange differences in the opinions of physiologists of late years, concerning the use and value of the serum-albumen for the nutrition and the maintenance of the functional activity of the animal organism.

In one case of chronic nephritis in which, notwithstanding the albuminuria and increasing dropsy, a really ravenous appetite existed (an exceptional case), and the regular process of digestion was only interrupted by an occasional diarrhœa, I noticed that the anæmia, emaciation, and loss of strength increased uninterruptedly. This fact should not cause astonishment when we bear in mind the enormous quantities of albumen that are eliminated with the urine in this disease.

For months at a time, as the figures given above prove, 10, 15, 17, and even more grammes of albumen (dry weight) may be excreted daily through the kidneys. The importance of this loss to the store of albumen in the blood-serum has been very clearly shown by Vogel.[1] When, as the disease progresses towards recovery (a rare event) or towards secondary atrophy, the daily loss of the albumen in the urine decreases, the appetite, it is true, returns; but we also invariably find that the general nutrition of the patient improves, his color becomes fresher, and his strength increases. When secondary atrophy of the kidneys sets in, the well-known hypertrophy of the left ventricle is developed, while at the same time the general nutrition improves; this lesion is invariably absent in the bodies of those who succumb at the height of the chronic nephritis. It is the consequence of the process of contraction.

I must explicitly assert here that my experience has convinced me of the correctness of the opinion expressed in another place, that chronic nephritis, when it does not lead to secondary atrophy of the kidney, does not as a rule cause hypertrophy of the left ventricle of the heart. This I do, because some doubts have been expressed as to the correctness of this opinion.

This assertion is based upon the results of the autopsies conducted by me both before and since my former publication on this subject saw the light.

If we accept Traube's theory that the hypertrophy of the left ventricle of the heart, which so constantly accompanies granular atrophy of the kidneys, is the result of the increased resistance in the arterial system caused by the atrophy of the kidneys, it may be claimed that the resistance afforded to the circulation by kidneys which are in a state of chronic inflammation is, perhaps, quite as great.

---

[1] L. c. S. 267.

The marked bloodlessness of kidneys that are in a state of chronic swelling favors this idea. In the production of the effect in question, however, both the quantity and the constitution of the blood contained in the vessels are of essential importance. In what has preceded I think I have demonstrated satisfactorily that in the course of chronic nephritis the blood-formation is invariably disturbed, and that anæmia, therefore, is the invariable consequence of this disease. Just as invariably, the blood which remains is diluted by the water retained in it in consequence of the renal malady, so that, even under perfectly normal blood-pressure, watery transudations take place through the walls of the vessels into the cavities of the body and into the interstices of the tissues. The result of this is that, in almost all cases of chronic nephritis, an increased tension in the aortic system, which, according to Traube's theory—the only tenable one in my opinion—is the sole cause of the cardiac hypertrophy, does not exist.

The pulse of these patients certainly does not exhibit that character on which Traube justly laid so much stress, and which had enabled him to detect the insidious renal malady, the granular atrophy, by the pulse alone. In a few exceptional cases, and even then only at the commencement of the chronic parenchymatous nephritis, we find the pulse for a short period full, tense, and jerking, and notably diminished in frequency; it is far more frequently soft and small, because the arteries are but sparingly filled. The heart, on the contrary, in most cases takes part in the general emaciation; there is no reason why it should be nourished extra (dilatation of the left ventricle has not been found a single time at the autopsy), and even if there were, the requisite material is lacking.

I have already shown that the kidneys, when in a state of chronic inflammation, are unable to perform properly their natural task—the removal of the excess of water from the blood; I have now to inquire how far, when in this condition, they may still be capable of performing their other important functions, the *depuration of the blood from the specific constituents of the urine.* In pursuing this inquiry, I will, for reasons already given, take into consideration practically only the most important constituent of the urine, the urea. It is evident, from the details already given of the results obtained in our hospital from the analyses of the urine passed by patients suffering from chronic inflammation of the kidneys, that the amount of urea excreted daily in this disease is far below what may be considered as the normal average excretion in healthy persons. One might expect then, that, if retention of urea in the blood and tissues be really the cause of uræmic attacks, such attacks would necessarily and invariably occur in the course of chronic nephritis. Nevertheless, uræmic attacks are by no means frequent in this disease, and only in exceedingly rare cases are they the immediate cause of death. It behooves us in this connection to remember that the production of urea in the human body does not by any means take place in fixed and immutable quantities; that, on the contrary, it depends, in the first place, upon the quantity of nitrogenous material present in the body and participating in the nutritive changes; in the second place, upon the quantity of nitrogenous food ingested and assimilated; and finally, in the third place, upon the degree of activity of the muscular and nervous systems. All these factors, which exert an influence on the production of urea, are, however, diminished in chronic inflammation of the kidneys. The great mass of the nitrogenous material in the body has dwindled away, the quantity of food taken and assimilated is reduced to a minimum, and the dropsy and the debility of the patients prevent muscular movements. It follows of necessity that the production of urea in these patients must be diminished and remain below the normal. Hence, we would not be justified in concluding that the retention of urea in the blood and tissues does not excite uræmic attacks, because these attacks are decidedly rare in chronic nephritis, in spite of the very scanty excretion of urea through the kidneys. For who can say that even the small amount of this substance excreted with the urine does not represent fully the quantity which is produced in the tissues?

But there is also another circumstance which may have some influence in preventing the occurrence of uræmic attacks in chronic nephritis, viz., dropsy. Small as may be the amount of solid constituents, and consequently also of urea, contained in the fluid effused into the subcutaneous cellular tissue, still a certain appreciable quantity is contained in it, and this quantity, as our investigations have shown, is increased to a very considerable percentage in the fluid effused into the serous cavities of the body. Professor Edlefsen found urea, as I have already stated, both in the dropsical fluid and in the watery contents of the various serous cavities. If, now, we take into consideration the immense quantities of these watery transudations from the blood and other juices of the body, which are apt to collect in the serous cavities of these patients, we will be obliged to admit that these cavities constitute a capacious reservoir for the storage of the pernicious material which cannot be excreted. The dropsy, in this respect, supplies, so to speak, a sort of natural compensation for the insufficiency of the renal functions; and the urea is stored up in a harmless state in the fluid contents of the serous cavities, and in the meshes of the subcutaneous cellular tissue. My own experience of the exceptional occurrence of uræmia in chronic nephritis tallies entirely with these views.

In my account of the case of the boy St., I stated that, in spite of the enormous anasarca caused by the nephritis, he retained a ravenous appetite. He excreted, on an average, ten grammes of urea daily. In the course of the last two months of his life, during which the dropsy persisted, but varied in degree, he was repeatedly attacked with the most severe uræmic convulsions. For days at a time one attack succeeded another, so that in the intervals the patient did not awake out of the coma. Nevertheless, a remission again set in, consciousness returned, and the lad immediately pounced with his wonted avidity upon the food given to him. At first he vomited up a part of what he ate, but the digestion soon regulated itself, and the patient then recovered his strength in a certain measure; after a short interval a fresh series of epileptic attacks again interrupted the improvement, and the last attack finally proved fatal. The undisturbed digestion of this patient, and the abundance of nourishment taken, kept up an active tissue-change, the products of which could not be excreted through the kidneys and stored away in the dropsical fluids in sufficient quantities to prevent a dangerous accumulation in the blood and tissues.

The importance of the dropsy as a reservoir for the pernicious urinary constituents appears to me to be established beyond question by Case VII. detailed above. In that case the most violent uræmic convulsions broke out after a profuse perspiration had been caused by a hot bath, and in that way a dropsy of considerable extent had been almost entirely reduced in a few hours.

With regard to the relations of the secondary inflammations of other organs and tissues to the renal disease under discussion, I have nothing to add to what has been already said. These inflammatory processes must be regarded as the results of the irritation of the tissues affected, and of the walls of their vessels, by the nutritive fluid which is contaminated with the constituents of the urine.

I trust that in the above I have succeeded in demonstrating the dependence of the other symptoms observed in chronic parenchymatous nephritis upon the disturbances of the renal functions. It still remains for me, however, to demonstrate the connection between the disturbances of function observed during life, and the anatomical changes which are found in the kidneys after death, or rather to prove that these changes necessarily entail those disturbances of function.

It will perhaps be possible to perform this task satisfactorily at some future time, when our knowledge of the pathological processes of which we are speaking will be more complete than it is at present. I need not hesitate to call attention to the vast gaps which exist at the present time in our knowledge of this special subject; they are clearly revealed by the contradictions which are found in the statements made by different authors in regard to purely objective histological lesions in the diseased kidneys. The expression made use of by one of our most deserving pathological anatomists: "The pathological anatomy of the inflammation of the kidneys is in truth the most extensively studied, but at the same time the least finished chapter in our department,"[1] is true even at the present day.

---

[1] *Rindfleisch*, Lehrbuch der pathologischen Gewebelehre. Leipzig. 1867–69. S. 416.

At all events, the condition of affairs is certainly not so simple in the chronic as it is in the acute parenchymatous nephritis. In the former the physiological consequences of the anatomical changes do not correspond in all respects with the results obtained from physiological experiments, as it is possible for them to do in the acute form. In the nature of things, physiological experiments can only disclose to the investigator the entire process which takes place in an affection running an acute course. Nevertheless, we are forced to base our reflections on the results of these experiments, which alone have thus far yielded any conclusive information upon the nature of inflammation. A portion, moreover, of the chronic cases of renal inflammation commence as an acute affection. The facts ascertained by clinical observation prove that the circulation of the blood through the kidneys must be affected in the same way in chronic as it is in acute inflammation; that in the one case as in the other a dilatation of the vessels and an alteration in the physiological properties of their walls form the actual point of departure and the essential nucleus of the changes in the structure and functions of the diseased organs. Does the long duration of the inflammatory process cause any further special change in the physiological properties of the walls of the vessels, or does it perhaps lead to perceptible alterations in their histological structure? If so, to what extent is this possible? These are questions which, it seems to me, have as yet received too little consideration, notwithstanding the zeal with which pathological anatomists have pursued the study of the inflammatory processes in the kidneys. At all events, even in chronic nephritis a complete arrest of the circulation through the kidneys never occurs; the nutritive changes and the functions of the diseased organs continue, though in an altered manner. Caseous degeneration of the diseased parts of the organs is never met with, and I have never yet witnessed a complete suppression of urine.

The facts elicited by clinical observations, however, compel us to admit that the physiological properties of the walls of the vessels are far more profoundly altered in the chronic than in the acute form of nephritis. This is evident not only from the long duration, not to say the permanence of the pathological condition, but also from the degree of the disturbance of the physiological functions.

My late colleague Colberg believed that he could positively confirm this assumption, as far as the functional vessels of the kidneys are concerned, by the anatomical demonstration of distinct structural changes in the glomeruli. In the kidneys of the often mentioned day-laborer K. he found glomeruli five times as large in the linear diameter as the glomeruli of normal kidneys. I remember quite well that he showed me these enlarged glomeruli in his microscopical preparations, and that I compared them with others from healthy kidneys, and found his statement to be completely confirmed.

At the same time I will distinctly state that I am by no means ignorant of the great differences in the sizes of the glomeruli that may obtain in one and the same healthy kidney.[1]

Among Colberg's papers I found a drawing of one of these preparations. It is a great satisfaction to me to have this opportunity of making more generally known the observations of this industrious and careful investigator, from whose drawings the woodcut Fig. 11 was copied. Fig. 12 represents a "proliferated" glomerulus (as Colberg describes it in a note attached to the drawing) from a kidney in a state of chronic inflammation, magnified to 350 diameters. I have already stated in another place, moreover, that Virchow had long ago discovered that in chronic nephritis the capillary coils of the glomeruli are broader and more opaque, their walls thickened, and their nuclei considerably increased in number. Colberg

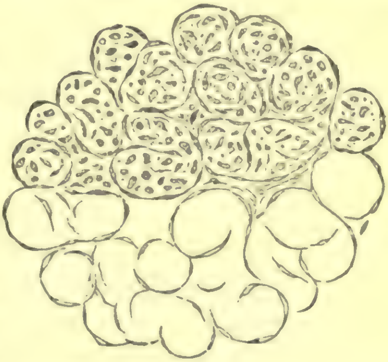


FIG. 12.
Proliferated glomerulus from chronic parenchymatous nephritis. × 350 diameters.

---

[1] The translator has preparations in his possession showing some range of size in the glomeruli derived from the same healthy kidney. The smallest are always situated high upon the cortical substance at the top of the stalk of the vasa ascendentia, the largest quite low down in the neutral zone; but in healthy kidneys the difference is inconsiderable, not amounting to two diameters.—R. S.

also evidently noticed the proliferation of the nuclei in the walls of the capillary tufts; this is proved by the upper half of the unfinished drawing, and by the term which he chose to describe the state of the glomerulus.

The dilatation of the blood-vessels, and especially of the tufts within the glomeruli, necessarily causes a retardation of the blood-stream, and hence also that diminution in the urinary secretion which is so constantly observed at the height of the disease, and often lasts for so long a time. The appearance, too, of albumen in the renal secretion is explained without difficulty by the alterations in the walls of the vessels, whereby their normal impermeability to this substance is abolished. We observe the same manifestations also at the commencement of acute parenchymatous nephritis. It is a striking fact, however, that the red blood-corpuscles, which are so commonly found in the urine during acute inflammation of the kidneys, are rarely found in this excretion in chronic nephritis. Perhaps the thickening of the walls of the Malpighian tufts may prevent the escape of the red corpuscles; the larger white blood-corpuscles, however, are still able to pass through them, and are often found in considerable numbers in the urine. This may possibly depend upon the well-known movement of the white corpuscles in the border-zone of the blood-stream. There are, however, other peculiarities of the urine excreted during chronic inflammation of the kidneys which demand a much greater degree of consideration from the observer, because they present a direct contradiction to the most widely accepted views concerning the physiology of the process of excretion in the kidneys, and also because they are not satisfactorily explained by the information we have obtained from experimentation concerning the alteration in the physiological properties of the blood-vessels in inflammation. The peculiarities to which I refer are, firstly, the excessive concentration of the urine, the specific gravity of which often largely exceeds that of the blood-serum from which it is excreted; and, secondly, the enormous percentage of albumen contained in it, which in particular instances can also for a time be considerably greater than the percentage of albumen in the blood-serum itself.

Years ago, while examining the urine of a dropsical patient with heart-disease—urine which, though free from albumen, was exceedingly scanty and had a specific gravity of 1040, I began to doubt the correctness of Ludwig's theory of the excretion of the urine, a theory still pretty generally accepted. I was forced to infer that the blood-serum of the dropsical patient must of necessity be much less dense than normal, and consequently must be much less dense than the urine excreted from this very serum.

According to Ludwig's hypothesis, the excretion of the urine by the human kidney takes place in the following way: in consequence of the pressure under which the blood in the glomeruli circulates, which is specially high because the arteria efferens has a smaller lumen than the arteria afferens, the water of the blood, together with the easily diffusible salts and other crystallizable substances, is forced out of the capillaries into Bowman's capsules. This process is one of simple filtration. The fact that the salts are not contained in the filtrate in the same proportions as in the blood-serum, is explained by the circumstance that the different saline substances transude through animal membranes with unequal rapidity, though exposed to the same degree of pressure. The fact that the albumen of the serum does not under the ordinary pressure pass through with the filtrate is also attributed to the nature of the filtering medium, for the vascular loops of the coils are not simple capillaries, but are covered on the outside by the epithelial layer of Bowman's capsule.

This filtrate, however, is not yet the perfected urine; it is subjected to another change by diffusion on its passage through the tubuli uriniferi. According to known laws of physics, an effort must be made to obtain an endosmotic equalization between the very watery fluid in the urinary tubules and the blood circulating in the capillaries surrounding them, which has been concentrated by the abstraction of this same watery fluid. Consequently, water must pass back again into the blood, and in compensation an equivalent portion of those solid constituents of the blood-serum, for which the separating membranes are permeable, will be added to the contents of the tubuli uriniferi.

Now, the slower the filtrate from the Malpighian tufts moves onward through the tubuli uriniferi, the more complete will be the endosmotic equalization between the two fluids that have been placed in this mutual relation; so much the more, consequently, will the degrees of concentration of the urine on the one hand and of the blood-serum on the other approximate to one another.

Observation demonstrates in fact two things: I. The dependence of the urinary excretion upon the blood-pressure in the entire aortic system, with the rise and fall of which the quantity of urine excreted coincidently increases or decreases. II. That the concentration of the excretion, *cæteris paribus* (the density of the blood-serum, for instance, being unchanged), becomes greater as the rapidity with which the excretion takes place diminishes, in an inverse ratio consequently to the slowness with which the filtrate from Bowman's capsules is forced onward through the renal tubules.

These facts all harmonize well enough with Ludwig's theory; their tenableness, however, has appeared to me much more dubious when I became convinced—by repeated direct comparisons of the specific gravity of the urine of a diabetic patient with that of his blood-serum, and also of the specific gravity of the urine of a man after six days of the sweat-cure with that of the serum of his blood—that it is possible for the urine to possess a higher specific gravity, consequently to be more concentrated, than the blood-serum out of which it has been excreted.

I still hesitated, however, to regard these clinical experiences as sufficient to disprove Ludwig's hypothesis. I persuaded myself that the conditions of endosmosis might suffer some modification from the quantity of albuminous substances contained in the blood-serum, because these substances, which are themselves prevented, by the nature of the intervening membranes, from taking part in the osmotic exchange, might possibly, by reason of their own strong affinity for water, interfere with the regular progression of this exchange by abstracting a larger quantity of water from the urinary fluid than would correspond to the endosmotic equivalent of solid matters given up to this fluid by the blood.

The characters of the urine, however, in many cases of chronic parenchymatous nephritis, show that Ludwig's hypothesis, even with the help of this supposition, is untenable. At the time the boy St. had his first attack of uræmic convulsions he was bled, and the specific gravity of his blood-serum was found by the pyknometer to be 1016.8. Unfortunately, no quantitative analysis of the albumen contained in the blood-serum was made, because it was employed for other researches. According to the results obtained from quantitative analyses in other cases, where the blood-serum was very watery, I do not think, however, that I would be justified in placing the quantity of albumen in this case at a higher figure than four per cent. Immediately before the outbreak of the convulsions, the boy had passed 65 c.c. of urine, which had a specific gravity of 1044, and contained fully five per cent. of albumen and 4.6 per cent of urea.

That under particular circumstances the urine should be denser than the blood-serum from which it is secreted, *i. e.*, should contain more solid constituents, is intelligible enough if we accept Bowman's theory of its secretion. That observer ascribed to the epithelium of the renal tubules just as specific an influence on the preparation of the excretion as is usually attributed to the special gland-cells of other secreting organs. The correctness of Bowman's theory would be fully proven if the results obtained by Heidenhain,[1] in his researches on the process by which the urine is excreted, should be confirmed. Heidenhain was led, by the results of these investigations, to accept Bowman's hypothesis that the special gland-cells continue to excrete the specific constituents of the urine, even when the excretion of water by the kidneys is completely suppressed.

According to this theory, the fluid filtered out of the Malpighian tufts is no more than the water which, in its passage through the tubes, is destined to wash out or lixiviate those specific constituents of the urine which the gland-cells of the tubuli have appropriated from the blood; it is of no importance for this theory whether these constituents be unaltered in form, or modified by the specific action of the cells.

If Bowman and Heidenhain describe correctly the process by which the excretion of urine is performed, and I am forced by my own clinical observations to believe that they do, then the secreting gland-cells of the kidney certainly do not forfeit their peculiar properties and power through inflammatory swelling; for the excretion furnished by inflamed kidneys may contain from four to five per cent. of urea. The quantity of water filtered out of the affected glomeruli is often exceedingly small, and hence, on its passage through the tubules, it becomes saturated with the products of glandular secretion to an unusual degree, and in this way the urine may attain a concentration far exceeding that of the blood-serum.

---

[1] Heidenhain. Versuche über der Vorgang der Harnabsonderung. *Pflueger's* Archiv. Bd. 4. S. 1.

This theory, however, does not by any means explain the second remarkable fact which we observe occasionally in examining the urine in chronic parenchymatous nephritis, namely, that the urine, under certain circumstances, indisputably contains a larger percentage of albumen than the blood-serum itself (see Case XVI.). If we regard the filtrate from the glomeruli of an inflamed kidney as analogous to an inflammatory exudation, as I believe we are perfectly justified in doing, we have reason to expect, according to our present knowledge of the composition of inflammatory effusions, that the percentage of albumen in it will always be lower, or at all events will never be higher than in the blood-serum, which has supplied it. If, now, it should happen that the urine contains a little higher percentage of albumen than the blood-serum from which it was excreted—and according to my own experience this unquestionably does occur—it can only be explained on the theory that water is withdrawn from the albuminous fluid after its escape from the glomeruli into the tubuli uriniferi, and during its passage through the latter. As to the manner in which this is effected, I have been able to form no decided opinion.

If the disease take a favorable turn, and a retrogression of the inflammatory process set in, the change must evidently be ushered in by a restoration of the normal properties of the walls of the vessels, for it manifests itself first of all by a remarkable alteration in the character of the excretion, from which we may conclude that the normal conditions of the circulation have been re-established. The quantity of the urine increases, and may, in especially favorable cases, rapidly overstep the normal average, so that, for the time being, much more water will be excreted by the kidneys than is taken into the system in the food and drink. Through the passages that are once more unobstructed pours (if we may be allowed to use the term) uninterruptedly the superabundant water from the blood-serum, which is constantly replaced by the reabsorption of the dropsical fluids. It is only by slow degrees that the walls of the capillary tufts of the glomeruli recover their physiological properties entirely. For a long time they continue to allow albumen to transude with the water and the salts, but in constantly decreasing proportions. Not until the recovery is complete does the albumen entirely disappear from the renal excretion, and this is invariably long after the dropsy has entirely vanished.

Only a few of the cases, however, in which the inflammation subsides and dropsy disappears, attain complete recovery.

Too much of the kidney substance may have been destroyed, under the influence of the inflammatory disturbance of nutrition; the channels left patent may be too narrow for the mass of blood which, as the general nutrition improves, fills the vessels, those of the kidneys among the rest; the pressure exerted upon the walls of the glomeruli that remain may be too great. Hence, under the abnormally elevated blood-pressure, which is still further increased by the consecutive hypertrophy of the left ventricle, the atrophied kidneys excrete a watery and constantly albuminous urine.

I have still a few words to add about the formation of the casts which are found in the urine in almost every case of chronic parenchymatous nephritis—often in enormous numbers.

I am convinced that the great mass of them—that is, all the hyaline ones—are produced simply by deposits of spontaneously coagulable albuminoid substances derived from the albuminous urine. This view is confirmed by the great richness of the urine in albuminous substances. As for the dark, granular casts, I willingly admit their development out of adherent epithelial cells that have undergone inflammatory degeneration, although in my microscopical examinations of the diseased kidneys I have never succeeded in finding any such adherent plug in the lumen of a renal tube, in lieu of its epithelial lining. Still, in kidneys which are the seat of chronic inflammation, the conditions requisite for the formation of such casts in the way just mentioned are certainly present. With regard to the broad, waxy, usually yellowish casts, I consider it established that they are formed principally in the straight tubes of the pyramids. I believe that the study of a preparation, presented to me by my colleague Heller, enables me personally to confirm the theory advanced by Rovida, that they are developed by a sort of excretion from the renal epithelium. These casts, as a rule, do not become at all numerous until the renal disease has lasted for a long time; but, when secondary atrophy of the kidneys has set in, they are sometimes found in the urine in extraordinary quantities. I have already in another place expressed a suspicion that this form of cast may be developed in those straight tubes whose radicles in the cortical substance have been either in part or entirely destroyed. It is possible that, in consequence of the cessation of their physiological functions, an essential alteration takes place in the nutrition of the epithelial cells, which may result in abnormal metamorphoses of their substance.

Duration of the Disease.

The duration of chronic parenchymatous nephritis differs very materially according to the cause by which each individual case is occasioned. In very many cases the renal disease must be regarded as a secondary affection, as the finishing stroke (Endkatastrophe) which hastens the fatal termination of some other chronic malady, and in such cases death may ensue after the expiration of a few months. In those cases in which the affection has attacked, as a more independent affection, individuals previously in good health, or has been produced by malaria, it is often impossible to determine the exact date at which it began, because the commencement of the disease was not attended by any particularly striking symptoms.

When once the malady has reached any height—if, for instance, dropsy has already supervened, we may confidently look forward to a protracted illness. It is only under particularly favorable conditions—*e. g.*, when a speedy removal from a malarious district is possible—that the affection can take a turn for the better at an early period.

The dropsy may last for years, and keep the patient all this time confined to his bed. Even when the dropsy disappears, the patients are not cured; they may drag out existence for a few years more, with the symptoms of secondary atrophy of the kidney, but finally succumb to the consequences of the nephritis.

But there are other cases in which the disease proves fatal, after the lapse of a few months, from dropsy or from secondary inflammation of other organs, or from uræmia.

Prognosis.

In considering the probable termination of a disease, the proper course is unquestionably first of all to ask oneself, *is this disease really curable or not?* In Bright's first publications on the diseased conditions of the kidneys which have since been called after him, this question was answered pretty decidedly in the negative.

In consequence of the great confusion of views which exist at the present day with regard to the affection of which we are treating, we need not wonder that the answers to this question which reach us from different physicians should be most contradictory. This confusion of views also makes it very difficult, if not actually impossible, to sift out from the literature of the subject enough serviceable material to enable us to give a conclusive answer to this question.

The physician who calls every case of albuminuria that he encounters "Bright's disease," and speaks of *chronic Bright's disease* or *chronic nephritis*, when the albuminuria has lasted for more than a few weeks, may correctly plume himself upon the number of cures he has effected. Those, on the other hand, who confine the term Bright's disease, or chronic nephritis, to the more distinctly marked cases that pursue a truly chronic course, attended by great anasarca, will scarcely give a more favorable prognosis for them than Bright himself did. I myself am of opinion that all hope of the possibility of complete recovery must be surrendered after the affection has lasted for a certain time.

Clinical experience certainly confirms the generally bad prognosis for these cases; even to one whose judgment has not been formed by practical experience, it must appear doubtful that vessels whose physiological properties have been perverted for months at a time, and whose walls have perhaps undergone great alterations in their histological structure, could ever again recover their natural condition. This latter supposition is not supported by analogously favorable observations in other organs which are more accessible to direct observation. The only point derived from experience which can be adduced in its favor is the fact that, after chronic inflammatory processes in the outer skin, such as chronic eczema and psoriasis, have lasted for years, a complete restoration of the affected parts of the skin to a normal state is still possible.

It would, however, be decidedly wrong to state to patients affected with chronic parenchymatous nephritis, that the disease invariably terminates fatally. I have already spoken of a case of scarlatinal nephritis, which I saw with my colleague Bockendahl, in which, after the obstinate dropsy had been removed, the albuminuria continued for eighteen months, and then perfect recovery set in. That patient is alive now, ten years after his sickness, and has grown to be a vigorous young man. An innkeeper, forty years of age, was confined to his bed for an entire year on account of extreme dropsy due to chronic nephritis, which had been caused by exposure to severe cold, but nevertheless he recovered completely. The very results of my anatomical examinations of the kidneys of persons who had apparently recovered after the dropsy had existed for a year or more, but whose urine remained albuminous until death was finally caused by the secondary atrophy of the kidneys, have convinced me that even in the more severe cases of parenchymatous nephritis we should not give up the hope of a possibility of recovery. For these examinations showed me that, although a very large part of the secreting structures of the kidneys was destroyed, yet those portions which remained had recovered perfectly their normal microscopical appearances. It may be—indeed, I am actually convinced, that all the urinary tubules of the cortical substance are not affected in the same degree by the chronic inflammatory process. No one, however, who has carefully studied microscopical preparations from the cortical substance of one of these large yellowish white kidneys, will be able to assert that he has found any considerable number of normal tubuli uriniferi supplied with a normal epithelial lining.

The prognosis of this complaint, however, would be far more favorable if it were recognized earlier than is unfortunately invariably the case. As a rule, attention is not drawn to the kidneys until the tell-tale dropsy has indicated the threatening danger; only then does the physician deem it necessary to make an examination of the urine, and he is apt to give his patient up for lost, when he finds that the fluid on the application of heat coagulates in the test-tube to a stiff jelly. The case of the nurse S., detailed on a preceding page (Case XVI.), shows by how many months the albuminuria may precede the dropsy, and how much time therefore may be gained for treatment before the dropsy sets in. This time gained will certainly not be useless in those cases in which it is possible to remove the noxious influence which has excited the renal disease. Experience teaches that a nephritis which has set in can be rapidly cured by the speedy arrest of a chronic suppurative process, *e. g.*, by the removal of a limb affected with fistulous openings into a joint (Bardleben[1]), or by bold incisions into peripleuritic abscesses with subsequent drainage (Bartels[2]); whereas, without the operations, according to all experience, the nephritis would unquestionably have pursued a chronic course and most likely would have proved fatal. Nor is the timely recognition of the nephritis which is caused by malaria of less importance. Here complete arrest of the paroxysms of fever by sufficiently large doses of quinine, given for a sufficient length of time, is the first requisite for the cure of the renal malady; above all, however, where it is possible, a speedy removal of the patient from the malarious district should be effected. Repeatedly have patients, who came from the west coast of our peninsula to be treated in our hospital here in Kiel, suffering not only from fever, but also from extreme albuminuria and commencing dropsy, recovered from their renal affection in a few weeks after the paroxysms of fever were stopped. When, however, the patients did not seek assistance soon after the dropsy appeared, a rapid recovery was in no instance attained, even in cases in which the paroxysms of fever had ceased long before the admission into the hospital.

---

[1] Lehrbuch der Chirurgie und Operationslehre. Berlin 1870. Bd. 1. S. 263.

[2] Ueber Peripleuritische Abscesse. Deutsches Archiv für klin. Medicin. Bd. 13. S. 21.

Nevertheless, even in fully developed cases in which the dropsy has existed for months, so long as the renal affection is not complicated by some incurable constitutional or organic disease, I do not by any means regard the treatment as impotent, provided only both the patient and physician employ the means of cure at their command with sufficient patience and persistence. I may, however, positively state that such cases never get well spontaneously.

The prognosis, consequently, will be different in the individual cases, for reasons entirely independent of the external circumstances surrounding the patients: in the first place, it will vary with the cause and the possible complications of the kidney disease connected therewith. It will be more favorable when the determining cause is one that can be removed; and, on the other hand, it will, of course, be absolutely bad when, as often happens, the renal disease is complicated by some incurable constitutional or organic malady. In the second place, in forming an opinion as to the probable issue of the affection, its duration must be considered. The longer it has lasted the less is the prospect of the treatment being successful. Lastly, the degree of the functional disturbance of the kidneys furnishes a gauge not only for the imminence of the immediate danger to life, but also for the extent of the anatomical changes which have taken place in the kidneys, and therefore for the prognosis in general. The most reliable information concerning the degree of the functional disturbance will be obtained from the examination of the urine. It may be regarded as favorable when the urine is passed in a nearly normal or in an excessive quantity, when it has a nearly normal specific gravity, and when the percentage of albumen does not exceed moderate bounds (1 to 2 per cent., for example). A favorable prognosis is also justifiable when the renal excretion after an antecedent reduction once more increases in amount, and the specific gravity and percentage of albumen become lower. A reduction of the quantity of urine to a few hundred cubic centimetres, an extremely high specific gravity (1035 to 1040), and the presence of three or four per cent. or even more of albumen, invariably betokens a high grade of pathological changes in the kidneys; there is then small prospect of a return to the normal condition, and the apprehension of a speedy fatal termination is well grounded.

Such marked disturbances of the renal functions are always associated either with commencing or advancing dropsy, and hence any serious amount of general dropsy in chronic nephritis is, as a rule, of evil omen. If the patient does not die during the continuance of the dropsy, it is apt, when it has once become excessive, to persist for a long time, and then a complete recovery is rarely attained. More frequently the dropsy subsides after a gradual increase in the quantity of water excreted by the kidneys; but the urine, though passed in larger quantities, exhibits already all the features which indicate the development of secondary atrophy of the kidney; it is pale, of low specific gravity, never even temporarily attaining a normal density, and contains albumen, although in smaller quantity than it did previously, and casts in extraordinary number.

Simultaneously the physical signs of hypertrophy of the left ventricle are developed: the area of præcordial dullness is increased; the heart's impulse becomes heaving; the aortic valves close with a sharp, distinct sound, and there is remarkable tension of the arterial pulse. After complete subsidence of the dropsy the patient, it is true, may gradually regain both weight and strength in some degree—enough, perhaps, to enable him to return to his usual avocation once more. He retains, however, his pallid look, his skin is flaccid, and sooner or later symptoms set in which must be ascribed either to the consecutive hypertrophy of the left ventricle, or to uræmic poisoning;

among those due to the former are a sense of oppression, palpitation, and vertigo; among those due to the latter, dyspepsia, intractable vomiting, neuralgia, cramps, and perhaps inflammatory effusions into the serous cavities.

In three cases I had the opportunity of following and observing the course of chronic parenchymatous nephritis from the very beginning of the dropsy until the fatal termination after the development of secondary atrophy. All three cases occurred in males aged respectively 18, 25, and 30 years. All three were excessively dropsical for a long time. In none of them, after the dropsy had finally vanished, was there the slightest reappearance of the œdema. Two of them lived two or three years after the disappearance of the dropsy. One took a long journey, resumed his occupation as a mining overseer, and died of pericarditis on the occasion of a visit to his parents. The second worked as a moulder in an iron foundry for two years after his apparent recovery, and then died of pneumonia. The third, however, remained so weak that he was unable to follow any avocation. He died with uræmic symptoms a year after his dropsy had entirely subsided. The history of this case (that of Herr Kr.) is given below. Detailed accounts of the condition of the urinary excretion in this case have already been given.

Case XVII.—Herr Ad. Kr., of Altona, aged thirty years, nine months before his admission into our hospital returned to Europe from the United States of America, where he had resided eight years. At first a merchant, he afterwards became a farmer in the State of Missouri, where he had repeated severe attacks of malarial fever. These attacks weakened him to such an extent that he resolved in October, 1866, to return to Europe. After his return home his condition did not improve, but, on the contrary, his lower extremities almost immediately began to swell, as well as his scrotum and abdomen, and his strength decreased still more.

On the 6th of July, 1867, he was admitted into our clinic. Although naturally a robust man, he was then exceedingly emaciated, his countenance was pale and cachectic; the lower extremities, scrotum, and abdomen were excessively swollen and dropsical to such a degree that on the thighs and abdomen the well-known distention marks in the corium existed. His subjective symptoms consisted principally in a feeling of general weakness, and in severe attacks of palpitation of the heart and oppression of breath. To determine the cause of these attacks a physical examination of the thorax was made, which showed that the diaphragm was pressed upwards to the level of the nipples by the ascites, while flatness on percussion on the left side below the scapula indicated a collection of fluid in the left pleural cavity. The area of præcordial dullness exceeded the normal limits somewhat, in the upward direction. The heart-sounds were clear. Pulse 120, small. Temperature normal.

In the first twenty-four hours 910 c.c. of urine were passed. It had a sp. gr. of 1021.5, and contained over two per cent. of albumen and a small number of casts, most of them small and hyaline.

An infusion of digitalis was ordered, but it produced no alteration in his condition beyond a reduction in the frequency of the pulse, and it was given up because the previously poor appetite became still worse under its use. It was especially remarked that the digitalis exerted no influence upon the excretion of urine. Indeed, this became still more scanty during the first week after the patient's admission, while the swelling of the subcutaneous areolar tissue extended and involved also the hands and the face.

On the 15th of July, pain on the left side of the thorax, and a loud pericardial friction sound situated about the apex of the heart, showed that pericarditis had set in. These symptoms disappeared, however, after a few days, and on the 20th of July the patient began a sweat-cure. Every day he remained for an hour in a warm bath (40° C.—104° F.—at least), and was subsequently wrapped up in blankets, in which he sweated profusely. This treatment certainly effected a considerable reduction in the dropsy, but the excretion of urine continued abnormally scanty. Before the beginning of September the average quantity of urine passed in twenty-four hours did not exceed 600 c.c. During this period the urine had a high specific gravity (on an average 1026), and contained rarely less than two per cent., and on some days even as much as three per cent. of albumen.

In the last few days of the month of August the patient complained several times of chilly feelings, and in the beginning of September lancinating pains in the left side and slight persistent fever set in. An examination revealed a loud pleuritic friction sound under the left scapula and in the left axillary region. The sweat-cure was immediately interrupted and gamboge ordered in pills.

In the early part of September an exudation took place into the right pleural sac, which reached nearly up to the ridge of the shoulder-blade behind. The excretion of urine remained as scanty as before, and the dropsy once more increased. During the months of October and November, when the daily quantity of urine fell to an average of 533 c.c., the dropsy was excessive, especially in the legs and scrotum. The specific gravity of the urine during this period was on an average 1027, but was subject to very great fluctuations, and the percentage of albumen diminished slightly. Once more an attempt was made to combat the increasing dropsy by hot baths, but it had to be discontinued on account of the difficulty of transporting the patient, who was now so swollen as to be perfectly helpless, out of his bed into the bath-tub and back again. From the end of October fifteen drops of oil of turpentine were administered four times daily, but without any effect either upon the urinary excretion or the dropsy. However, the exudation in the right pleural cavity diminished and was gradually entirely reabsorbed, but at the same time sharp pains set in, in the hepatic region. These pains were undoubtedly due to a perihepatitis, for with the stethoscope a loud friction sound synchronous with the respiratory movements could be heard over the entire region of the liver, extending even below the arch of the ribs on the right side. This loud friction sound remained noticeable nearly throughout the whole month of November. From the end of November the patient took a decoction of colocynth, which produced three or four watery stools daily, but caused no improvement in his general condition. On the contrary, the patient felt much exhausted, and lost his appetite almost completely; for this reason we were soon compelled to discontinue the drug. In the early part of November small vesicles appeared on the skin of both thighs, and after these burst the epidermis became detached over extensive areas. From this time forth the dropsical fluid oozed out in such quantities from the denuded corium, that the bed was soaked through and through, and considerable quantities of the fluid could be collected in vessels placed beneath the bed. This at all events had the effect of reducing the swelling of the previously enormously swollen legs. In the course of the month of December the fluid ceased to trickle out, and the excoriated and excessively painful spots became once more covered with skin. At the same time the urinary excretion increased to a daily average of 770 c.c. In January, 1868, the swelling of the legs again increased to such an extent that fresh rents took place in the epidermic covering of the thighs, and once more enormous quantities of fluid trickled away. Meanwhile the quantity of urine excreted remained unchanged. The swelling of the legs rapidly subsided, and on January 20th the draining off of the dropsical fluid ceased.

On the 23d of January he had a severe rigor followed by fever, the temperature reaching 40° C. (104° Fahr.), and the pulse 160. Severe pains were experienced in the cardiac region, which were explained by a loud friction sound, heard in the region of the apex of the heart with the stethoscope; the sound was synchronous both with the pulsations of the heart and the respiratory movements; ordered quinine with digitalis.

The fever lasted for five days, when defervescence set in almost suddenly. On the 31st of January the friction sounds had not yet entirely disappeared; but the general condition of the patient was better than it had been at any time since he came under observation.

From this time forward the patient's appetite, which hitherto had always been poor, improved as did also his strength, and the quantity of urine passed daily gradually increased. On the 28th of January, during the febrile attack, a spontaneous perspiration set in for the first time since his admission, and it was subsequently repeated almost every day. The dropsy diminished more and more. In March the quantity of urine increased to a daily average of 950 c.c., and in April to 1120 c.c.; at the same time the specific gravity fell respectively to 1020 and 1018, and the percentages of albumen to 0.59 per cent. and 0.64 per cent. on the average.

During the warm summer months of 1868 the dropsy entirely disappeared, and Herr Kr. was able to be nearly the whole day in the open air, and even felt himself strong enough to take short walks. In the beginning of August, however, a severe diarrhœa set in suddenly and without apparent cause, and at the same time a marked diminution in the excretion of urine took place (on one day only 400 c.c. were passed). The diarrhœa was quickly subdued, but the ordinary excretion continued scanty, the average daily excretion during the month being under 670 c.c. The specific gravity rose during this time to 1024, and the percentage of albumen was often considerably over one per cent.

When, in the beginning of October, I returned home from a long vacation trip, I found Herr Kr. once more excessively swollen over the entire body, and the abdominal cavity greatly distended with fluid. The patient complained of severe pain in the bowels, and the abdomen, especially in the region of the umbilicus, was so sensitive to pressure that he could not bear the slightest percussion. With all this his general condition was not very bad, and even his appetite was pretty good. Iodide of potassium was ordered, but it produced no alteration in his condition. However, the quantity of urine increased slightly.

Towards the middle of December protracted hot baths were once more prescribed, and this time they very soon caused not only an increase in the quantity of urine, but also a rapid disappearance of the dropsy. Whereas in December the average quantity of urine passed daily was 890 c.c., in January it increased to 1100 c.c., in February to 1200 c.c., and in March to 1280 c.c. At the same time the specific gravity and the percentage of albumen fell. On the 31st of January the hydrops anasarca had entirely disappeared. In the abdominal cavity, however, which remained very sensitive to pressure, a fluid (peritonitic) exudation could still be demonstrated.

Now, for the first time, the extreme degree to which the patient was emaciated became apparent. He complained too of great weakness, and suffered for a long time from excessively severe headaches, which could only be relieved by ice-bags and morphia injections. Iodide of iron was prescribed. At the end of February every trace of dropsy had disappeared, and the warm baths were discontinued.

On the 3d of June, 1869, after having been a year and eleven months in the hospital, Herr Kr. was discharged; he had improved greatly, and in the preceding six weeks had gained 5¾ lbs. in weight. He still complained frequently of headache, and for some time previously his sight had been somewhat affected, in explanation of which we had discovered a whitish cloudiness and swelling of the papillæ of the optic nerves (œdema).

During May he had passed on an average 1165 c.c. of urine daily, the average specific gravity of which was 1014, and the average percentage of albumen 0.444 per cent. An examination of his heart made before he left gave the following results: the apex-beat not distinctly perceptible, area of præcordial dullness of normal limits, heart-sounds muffled, the diastolic sounds over the aortic valves rather louder than the second sound over the pulmonary artery. Pulse 92, and not remarkably tense.

Herr Kr. went into the country to some of his relations to follow a milk-cure. At first the country suited him very well; he gained strength, and within two months added 8 lbs. to his weight.

In August, however, he began to suffer from attacks of palpitation and severe dyspnœa, which obliged him to come back to the hospital on August 17th. On his arrival he again looked very pale, and could walk only with difficulty and with the help of a stick. Respiration quick. The physical examination of the chest revealed considerable distention of the pericardium with fluid effusion; the dullness extended upward to the cartilage of the second rib on the left side one centimetre beyond the left nipple, and on the right side as far as the right nipple. Heart's beat imperceptible; between the left border of the sternum and the left nipple we could distinguish, however, an indistinct tremor. Heart-sounds faint and muffled. Pulse quickened and tense. Ordered digitalis. The next day there was cough and pneumonic expectoration. In the upper lobe of the left lung a pneumonic infiltration was discovered. At the same time frequent vomiting set in, which obliged us to discontinue the digitalis. The urine was very scanty, and contained albumen and casts in remarkable abundance.

Unfortunately for the first few weeks of this period of the observation (it was vacation time), no thorough examinations of the urine were made.

In the course of a week the pneumonia began to subside, but the patient did not rally, because the vomiting persisted obstinately. It yielded to no remedy, and for a long time almost completely prevented the assimilation of food. It came on after every meal and caused the rejection of the greatest part of what had been ingested, both solids and fluids. In consequence of this the patient's strength failed him more and more; from the time he returned to the hospital he had never been able to quit his bed.

Towards the end of September the vomiting became less frequent, and was restricted to a single evacuation of his stomach in the evening; finally, we suceeded in arresting it entirely by a timely injection of morphine. This was, however, frequently followed by the regurgitation of a watery fluid in the morning after awaking; this fluid had a faintly acid reaction, and was sometimes very abundant. From this time forward he was once more able to take some roast meat and white bread.

Meanwhile the effusion in the pericardium had been reabsorbed, and in the beginning of October the area of præcordial dullness was but little beyond its normal limits, while the heart's sounds were loud and clear. But now other very troublesome symptoms set in. His sleep at night was very frequently broken by spasmodic movements of the entire body. To this was added a terribly annoying itching of the skin, which finally almost entirely deprived him of all rest, leaving his sensorium heavy and dull during the day-time. Under these troubles our patient's frame of mind was reduced to a state of desperation, he impatiently demanded his discharge, and on the 21st of December, 1869, he was taken to Altona by his friends.

During the last four months of his second sojourn in the hospital the quantity of urine passed by Herr Kr. had steadily increased. The specific gravity as well as the percentage of albumen, on the contrary, had become lower. In September he passed daily, on an average, 1,072 c.c. of urine, sp. gr. 1010, albumen 0.285 per cent.; in October, 1,313 c.c., sp. gr. 1010, albumen 0.317 per cent.; in November, 1,516 c.c., sp. gr. 1009, albumen 0.198 per cent.; in December, up to the day he left the hospital, 2,020 c.c., sp. gr. 1008, albumen 0.185 per cent.

Throughout the whole of this second period of observation, one remarkable feature was the extraordinary quantity of casts contained in the urine. Every day they settled down to the bottom of the urine vessel in the form of a copious grayish white sediment, while on the other hand, at the date of his first discharge in May, only very few casts were contained in his urine. In May they were mostly small and hyaline, but a few months later the dark granular and the broad wax-like casts decidedly predominated. On the addition of iodine both the broad and the dark granular casts were simply colored yellow. When he left the hospital no trace of dropsy existed. I am indebted to Dr. Auerbach, of Altona, for the following details of the subsequent course and termination of this remarkable case. Dr. Auerbach wrote to me on the 30th of January, 1870, to the following effect:

"Last week I saw Herr Kr. again. I found him very emaciated and anæmic, his extremities were moderately anasarcous, and his skin was dry and branny and terribly scratched, particularly on the face and trunk. The area of præcordial dullness was increased, the diastolic aortic sound very loud, pulse 88 and tense, respiration free. While I was holding his hand I felt a convulsive twitching of his muscles. For the last eight days he has again been suffering from obstinate vomiting, and has repeatedly perspired very abundantly. The excretion of urine is much diminished, 500 c.c. in the twenty-four hours. The urine is very pale, its sp. gr. is 1012, and the percentage of albumen I estimate to be rather more abundant than during the last part of his stay in Kiel. His sensorium is so much affected that he does not recognize me (up to the beginning of 1870 Dr. Auerbach had been assistant physician in the medical clinic at Kiel). While in a hot bath which I had ordered he was seized with a well-marked epileptic attack, and died in profound coma on the evening of January 29th."

Dr. Auerbach also performed the autopsy, and sent me the following account of it:

"*Lungs* bound to the thoracic wall by easily-torn adhesions, but otherwise normal and containing air.

"*Pericardium* contained a considerable quantity of clear serum; both parietal and visceral layers covered with grayish-white deposits (the so-called tendinous spots?). Heart very large, left ventricle dilated, and its walls very considerably thickened.

"*Abdominal cavity* contained about 500 c.c. of clear, light colored fluid.

"*Intestines* greatly distended with gas, their serous coat presented a slate-gray color, and was attached by numerous bands of firm adhesions to the abdominal parietes. One especially firm pseudo-ligament took its rise from the middle of the transverse colon and was inserted into the abdominal wall opposite the fundus of the bladder. The coils of intestines were slightly adherent to each other.

"*Stomach* collapsed; mucous membrane much swollen, and bright red in color; small ecchymoses were scattered all over its surface, but there were no ulcerations, and no cicatrices. Pylorus free. The intestines were not opened.

"*Liver* small; its upper surface was completely agglutinated to the diaphragm.

"*Spleen* apparently of normal dimensions."

Dr. Auerbach sent the kidneys to me in Kiel, where I examined them. Both kidneys were small, and the capsules were firmly adherent, so that it was difficult to tear them off. The surface of both was knobby and uneven, granulated; the color dark brown. On the surface were scattered a few cysts, about the size of millet-seeds, filled with clear contents. One kidney weighed 80 grammes, the other 82 grammes. The first was 9 cm. long, 5 cm. broad, and 2.5 cm. thick; the second was 10 cm. long, 5 cm. broad, and 2.5 cm. thick. Upon section it was evident that the diminution in the size of the organs was caused essentially by atrophy of the cortical substance. The pyramids were much less affected by the atrophy. The microscopic examination showed a diffuse increase of the interstitial connective tissue, pretty evenly distributed throughout both kidneys; in several places the interstitial connective tissue had developed into thick fibrous bands, between which no trace of the true glandular tissue was any longer visible. In other parts, in the midst of these fibrous bands, round, dark bodies were found, which were surrounded by concentric layers of long spindle cells, and could still be readily recognized as atrophied Malpighian corpuscles. In other places, again, I found groups of perfectly preserved tubuli uriniferi, with normal Bowman's capsules and capillary tufts.

This case may be adduced as proof that cases occur in which the different stages of the so-called Bright's Disease, as taught by most authors, actually follow one another in regular order. Had Frerichs, in his work on Bright's Disease, detailed a case like that of Herr Kr., the reproach cast at him by Samuel Wilks, and quoted on page 324, would not have been justified. From my knowledge of the literature of this disease, however, I am certain that not many cases have been accurately observed and published in which the symptoms of the disease were developed in the same order as in the case of Herr Kr., and in which similar appearances were found at the post-mortem examination.

I must confess, however, that my investigations of the anatomical changes in this case have left me still in uncertainty as to whether I had to deal simply with a case of secondary atrophy of the kidneys, or with a complicated process.

The following occasional symptoms or accidents demand special consideration in connection with the prognosis: Œdema of the glottis and of the lungs is fraught with immediate danger to life; but still I have seen a first attack of pulmonary œdema recovered from. Pneumonic infiltrations of the pulmonary tissue have in my experience almost invariably terminated fatally when complicating parenchymatous nephritis. Phlegmonous inflammations of the subcutaneous cellular tissue, as a rule, have a similarly unfortunate issue; they terminate in gangrene, and the absorption of the gangrenous ichor is the immediate cause of death. Still, I have in two cases, in which gangrenous destruction of the scrotum took place, witnessed a complete recovery from the local lesion. Inflammatory exudations into the serous sacs may be completely reabsorbed. In the body of Herr Kr. we discovered unmistakable evidences of previous pleurisy, pericarditis, and peritonitis. The inflammations in these serous cavities were recognized during the life of the patient, and the existence of the exudations was demonstrated by physical examination. Uræmic symptoms of the most severe type may be recovered from, just as in acute nephritis, and complete recovery may subsequently set in; it is only after secondary atrophy of the kidneys has developed that they assume the almost absolutely fatal character which they possess in all atrophic conditions of the organs.

Diagnosis.

The practitioner who would recognize the above described dangerous renal affection early enough to justify the hope of being able to treat it successfully must make it a strict rule of practice to examine with assiduity the urine in all cases of those diseases which, as we know from experience, are liable to be followed by nephritis.

The diagnosis of chronic parenchymatous nephritis is not difficult when we have to deal with persons who present a pale and anæmic aspect, either with or without apparent reason, who are dropsical, and who pass but little urine of high specific gravity, containing a high percentage of albumen and numerous casts of various kinds. Nevertheless, the differentiation of this affection from other diseases of the kidneys also accompanied by albuminuria may sometimes, throughout every stage of its progress, be attended by certain difficulties. It is impossible in any given case to decide beforehand whether an affection of the kidneys that has set in with symptoms of acute nephritis will or will not become chronic.

Nevertheless, for all cases of nephritis following diphtheria, cholera, and the acute exanthemata, we can almost positively predict an acute course and a short duration. On the other hand, all those cases of nephritis which are due to exposure to cold, or which are developed during an attack of acute articular rheumatism, or during pregnancy, have a far more decided tendency to pursue a chronic course, as I have already remarked in another place. Even those cases which, arising under the last-named circumstances, commence with violent symptoms, such as copious renal hemorrhages, consequently in the same way as the graver forms of scarlatinal nephritis begin, may develop into chronic disease of the kidneys. My experience in several cases has convinced me of the remarkable fact that a nephritis which commences with hæmaturia, either during the course of a febris intermittens or soon after its disappearance, will usually quickly and easily yield to treatment; whereas, on the contrary, a nephritis which, under the same circumstances, begins insidiously, so that it does not attract attention until a marked degree of dropsy is developed, is especially obstinate. This is again a warning to us physicians of the necessity of constant watchfulness over the renal secretion in all malarial diseases.

In general, the diagnosis of a recent inflammation of the kidneys is justifiable when the urine is strongly tinged with blood, and presents the other peculiarities of an excretion furnished by inflamed organs. It is necessary to remember, however, that intercurrent hæmaturia may occur in the course of chronic nephritis, and even when genuine atrophy of the kidneys has taken place.

When both blood and epithelial cells are absent, while the urine is scanty, has a high specific gravity, contains a large percentage of albumen, and deposits a sediment composed of masses of detritus and very numerous casts, a large portion of which are dark and granular, the case is quite certainly one of chronic nephritis. Urine with a specific gravity of over 1030, and containing three or four per cent. of albumen, is of a certainty very rarely met with in acute nephritis.

In the course, however, of both the acute and the chronic forms of parenchymatous nephritis, conditions may arise which may render it next to impossible to distinguish the one form from the other, either by the analysis of the urine or by the rest of the symptoms which are presented. In such cases, therefore, nothing but further observation can enable us to decide which form of disease we have before us. In acute nephritis the dropsy is much more frequently entirely absent than in the chronic form, in which it is an almost constant symptom. Like chronic inflammation of the kidneys, amyloid degeneration of the organs causes albuminuria, and, as a rule, dropsy; and in many instances, moreover, the etiological influences are the same for both affections. Like chronic nephritis, amyloid degeneration of the kidneys very frequently occurs as a sequence of chronic suppurative processes of any sort, as the expression of the general cachexia, and also in syphilitic subjects who have become cachectic. In such cases it not unfrequently happens that we meet with both chronic parenchymatous inflammation and amyloid degeneration of the vessels in the kidneys of the same body. As far as concerns myself personally, I must admit my inability, with my present knowledge, to diagnose positively this combination during life.

For the separate and uncombined cases, however, I may lay down the following differential points:

In chronic nephritis the dropsy is more constant, and attains a higher grade. The urine is, at least for a time, passed in abnormally small quantities, and has then an abnormally high specific gravity and a dark color, is cloudy, and contains large percentages of albumen and numerous casts in the sediment.

In simple *amyloid degeneration* the quantity of urine passed is, as a rule, normal or abundant; it is either pale, or at least light colored, clear; as a rule, it has an unnaturally low specific gravity, and seldom contains, at the outside, more than one per cent. of albumen. A few hyaline casts may be found in the sediment, but occasionally none at all can be discovered. When chronic nephritis is in process of recovery, or in progress towards the stage of secondary atrophy, the urine may, in the first case temporarily, in the second permanently, present the same characters, in many respects, as in amyloid disease; in such cases, however (even in the convalescing ones, so long as the urine remains albuminous), we invariably find large numbers of casts in the sediment—enormous numbers, in fact, when atrophy has been developed. The patients in these cases, moreover, have in every instance suffered from high grades of dropsy, which was induced and maintained by a remarkable diminution of the urinary secretion. Further, hypertrophy of the left ventricle of the heart is superadded to the secondary atrophy of the kidneys, a complication I have never yet met with in connection with simple amyloid degeneration of the organs.[1]

Chronic nephritis can only be confounded in its terminal stage of secondary atrophy with the genuine contracted kidney—granular atrophy or cirrhosis of the kidney. This mistake is furthered by the great similarity of the characters of the urine in both diseases—pale color, low specific gravity, small percentage of albumen. In cases of genuine contracted kidney, however, the quantity of urine passed is generally far in excess of what is normal—a thing which in cases of secondary contraction after nephritis I have never witnessed. In the latter the sediment contains vast numbers of casts of every variety, whereas in the former only very few hyaline casts are to be found. Both conditions, in the great majority of cases, are accompanied by consecutive hypertrophy of the left ventricle. In cases of nephritis, however, extensive and obstinate dropsy, accompanied by the repeatedly mentioned diminution in the excretion of urine, will have invariably preceded the process of contraction, and have only subsided upon its establishment. In genuine atrophy of the kidneys dropsy is, in the majority of the cases, entirely wanting. In some cases a slight degree of dropsy appears every now and then in the course of the disease, as a fleeting and temporary symptom, or presents itself as a terminal symptom in patients who have meanwhile become marantic.

---

[1] With regard to the connection between amyloid degeneration and atrophy of the kidneys, two conditions which are occasionally combined, I will express my views hereafter

The development of dropsy even in these cases is wont to be associated with a notable diminution in the quantity of urine excreted. An important point, however, which serves to distinguish this condition from nephritis, is the fact that the urine, though scanty, as a rule retains its low specific gravity.

Even when, in the earlier stages of the process of genuine atrophy, during which dropsy is exceedingly rare, the daily quantity of urine passed becomes very small, and a transitory œdema is developed, the specific gravity of the urine never attains the extreme heights which are so commonly attained in chronic nephritis, at the commencement and during the increase of the anasarca.

The functional disturbances of the kidneys, caused by the disturbances in the circulation dependent upon severe cardiac lesions, can never be mistaken for nephritis, although under their influence the urinary excretion may be reduced to a minimum, and a marked degree of dropsy is wont to develop.

The general cyanosis which accompanies the so-called cyanotic induration of the kidneys, and the demonstration of its cause, prevent any such mistake being made. Moreover, the mode in which the dropsy spreads is characteristic; it is confined principally, as is well known, to the region of the inferior cava, and the upper parts of the body are, as a rule, entirely free from anasarca.

The urine excreted by kidneys which are in a state of cyanotic induration may be as scanty in quantity, have as abnormally high a specific gravity, and be as dark colored as the urine in the severest cases of chronic nephritis. It may contain albumen, too, but never in such immense quantities as are usually met with in the urine of chronic nephritis. The casts are few in number, and are invariably small and hyaline.

Treatment.

The treatment of chronic parenchymatous nephritis must be conducted, in general, upon the same principles as the treatment of the acute form of the disease. Above all, the physician must endeavor to obviate any existing injurious influences which are capable of causing the disease of the kidneys, or of favoring its prolongation. As a measure that may be adopted for this purpose, I have already mentioned the cure of chronic suppurative processes by operative treatment, and have adduced examples of the good results which follow this treatment. When the nephritis is a consequence of intermittent fever, the latter must be checked rapidly and thoroughly, and the patients, if possible, withdrawn from the influence of the malaria by removal from the miasmatic district. When syphilis exists, we should try to eradicate it from the system by the energetic employment of mercurials or of iodide of potassium, according to the indications of the particular case. When we are unable to fulfil the etiological indication, the prospects of a successful result to the treatment are poor. In many cases, however, the etiological moment is not discoverable, and we can obtain no indications for treatment from this source. These are just the cases in which the disease is often not discovered until it has made considerable progress, and which are often enough only brought to the attention of the physician after dropsy has been in existence for some length of time. It is necessary then to attack the disease by direct treatment. In the search after a satisfactory means of treating chronic nephritis, physicians have groped about and experimented even more than in the acute form of the disease. Bloodletting, laxatives, the so-called alteratives, all the diuretics and diaphoretics, and all the drugs that contain tannic acid, have been in turn tried and recommended, and at last we have returned almost to the identical point on which Bright stood at the time of his first publication, when he regarded all chronic renal diseases that are accompanied by albuminuria as incurable. These experiments, however, have at least brought us a negative profit, for they warn us against the useless employment of all medicines and modes of treatment that are directly weakening, or that derange the digestive organs of the patients, who are usually already anæmic. A special predilection has been shown for the various diuretics, which are constantly employed and recommended—most likely because dropsy, when due to other causes, is often successfully combated by them. With the exception of cantharides and squill, I have tried them all, even the scoparium spartium, which was recommended by Bright, and is a favorite home-remedy with people in this country, but I cannot say that I obtained any noticeable results from them. A medical man from Dittmarsch recommended to me the oil of turpentine as an excellent remedy for the renal dropsy that follows ague. In the chronic cases on which I tried it it did no good; in the acute cases it is superfluous, for these get well without it, when the directions given above are carefully followed. I am, consequently, disposed to doubt the curative effects of the oil of turpentine. Immermann[1] has recently strongly advised the employment of the diuretic salts, especially acetate of potash in large doses (five to ten grammes *pro die*), not only in all cases of acute Bright's disease, but in chronic parenchymatous nephritis of recent date, as well as in the relapses and aggravations of the latter. Immermann was astonished at the rapidity with which, in a number of cases of genuine parenchymatous nephritis, some of which were of long standing, all the symptoms of the disease were relieved and a cure attained by methodical diuresis. For the relatively recent cases I consider Immermann's recommendation justified, although I have certainly not always obtained such favorable results as he did. In cases of long standing the diuretic salts do little or no good.

The drugs which contain tannic acid were tried by Bright, and, especially since Frerichs recommended them, have been very generally employed. I have tried them in a variety of forms, and persisted in their employment for long periods, but was never able to satisfy myself that they produced the slightest effect.

The preparations of iodine, which we know are very rapidly excreted by the kidneys, have also been recommended as remedies for chronic nephritis. I do not doubt that the administration of iodide of potassium may exert a favorable influence on those cases of nephritis which depend on chronic suppuration; experienced surgeons have assured me of this, and I can even confirm it from my own experience. I am unable, however, to explain the physiological reason for this sanative action.

---

[1] Correspondenz-Blatt für schweizer Aerzte. Juni. 1873. No. 11.

In other cases of nephritis the iodide of potassium has proved entirely useless in my hands; in fact, in a bad case of hereditary syphilis of the skin, the inflammation of the kidneys actually set in while the patient was taking this salt, and terminated fatally in spite of the continued use of the drug. I must consequently state positively that of all the remedies which are said to act directly through the blood upon the renal tissue, not one has proved itself to be a reliable remedy against the chronic parenchymatous nephritis. Under these circumstances medical men long ago found themselves compelled to have recourse to other measures of treatment; some sought to combat and subdue the renal affection in some other way, while others were animated only by the hope of relieving the more insupportable symptoms of the disease.

The old opinion that chronic nephritis can be cured by local bloodletting, issues, setons, etc, in the lumbar regions, may be considered nowadays as entirely discarded. That the malady was never cured in this way is evident from the records of the older writers; that, on the other hand, all these measures were not merely useless, but actually prejudicial in this disease, will not be denied by any unprejudiced person.

I cannot speak much more favorably of the treatment of chronic nephritis by drastic purgatives. Even should these prove of any service in reducing the dropsy, the good that they would effect in this way would be far outweighed by the injurious influence they would exert on the general condition of the already anæmic patients.

Any one who bears in mind the fact that spontaneous diarrhœa so often occurs in chronic nephritis, and who remembers how quickly the patient's powers are weakened by it, and how difficult it is to check it, will certainly admit that the dislike of most physicians for the treatment of this affection by violent purgation is fully justified.

I have never known either the spontaneous or the artificially provoked diarrhœa to exert a favorable influence on the course of the renal disease; on the contrary, I look upon spontaneous diarrhœa as an exceedingly ominous symptom. Upon the outbreak of the diarrhœa the previously scanty urine invariably becomes still more scanty, its specific gravity rises, and the percentage of albumen increases. I have, moreover, never known the urinary excretion, after the diarrhœa had ceased, to become more abundant than it was before the watery discharges from the intestines set in; consequently there can be no reason to assume that the diarrhœa exerts any favorable after-influence on the condition and the functional capacity of the kidneys.

Here, again, the methodical employment of diaphoresis constitutes in my opinion the most reliable means of reducing the troublesome and dangerous dropsy; it is also the only treatment from which I believe myself justified in expecting a curative action on the process of disease in the kidneys, similar to that which it exerts on the acute parenchymatous inflammation of the organs. We must remember, however, that in the chronic form of the affection we have to deal with a much more extensive alteration both in the vessels and the tissues, and that consequently we ought not to expect from any mode of treatment a rapid restoration of the normal condition and the normal functions. If we wish to effect a cure by diaphoresis we must set to work in a thoroughly methodical manner and carry out the treatment with obstinate pertinacity.

Even Bright was to a certain extent aware of the value of diaphoresis. Among the remedies in the acute form of his disease of the kidneys he regarded[1] abstraction of blood as the most important, but admitted that it was entirely insufficient unless reinforced by purgation and by increased activity of the skin at the same time. He held strict confinement to bed to be the most reliable of all the methods at our command for the maintenance of the proper degree of activity of the skin, and without this he thought there was no prospect of curing the disease in our climate. At a later period he added that he should expect the most beneficial effects, even in chronic cases, from a complete change of climate. A voyage to the West Indies, and a residence on one of the healthier islands there, often effects a great change in the constitution, principally on account of the action upon the pores of the skin. I have not hesitated, therefore, to keep patients who were affected with chronic nephritis in bed, not only while the dropsy lasted, which in itself prevented free movement, but as long as the character of the urine showed that the process of inflammation still continued in the kidneys—as long, consequently, as albumen was contained in this excretion in large quantities. I have confined them to bed for very long periods, and only suffered them to leave it on hot days in summer. As soon as the weather became colder, or the quantity of urine diminished and the albumen increased, the patients were sent back to bed again.

---

[1] Cases and observations illustrative of renal disease. Guy's Hospital Reports, April, 1840, p. 160.

Of the patients who rigorously complied with these directions, and at the same time underwent the diaphoretic plan of treatment otherwise prescribed, some recovered completely, although at the time the treatment was begun they had already been excessively dropsical for months. The other measures for procuring diaphoresis, whether they consist in hot-air baths, simple hot baths, or in packing, must be persisted in for the same length of time as the confinement to bed. I have nothing further to add concerning the diaphoretic treatment to what I have already said while discussing the treatment of acute nephritis, except that in the chronic affection the diaphoresis must be continued without interruption until the urine shows that the inflammatory process in the kidneys has subsided. It may take years to attain this point if the malady has taken deep root. I have done this, and my results show that the thing is feasible, and that cures may even then sometimes be attained.

It is well known that patients who are suffering from parenchymatous nephritis scarcely ever perspire spontaneously; the skin has invariably a harsh, dry feel, and we do not always succeed at once in bringing on a perspiration by the ordinary diaphoretic measures. It was a noticeable fact, however, that persons who at the commencement of the treatment could hardly be made to sweat by a hot bath of an hour's duration at a temperature of 40° C. (104° F.), at a later period, after profuse perspirations had been produced daily by the hot baths, would perspire profusely on the application of a simple pack.

In chronic parenchymatous nephritis also I have repeatedly found, after the adoption of a methodical diaphoretic treatment, that, as soon as I succeeded in producing a profuse sweat every day, a more abundant excretion of urine set in, and that the percentage of albumen at the same time became reduced. Rosenstein also has laid stress upon the effect of diaphoresis in increasing the urinary secretion. In this fact it seems to me we have evidence not only of the symptomatic, but also of the curative value of diaphoresis in the treatment of chronic nephritis. I have already intimated my opinion as to the manner in which diaphoresis acts on the pathological condition of the kidneys. I believe that, by the long continued and daily repeated hyperæmia of the capillaries of the skin, the vessels of the internal organs — consequently those also of the kidneys — are relieved of the excessive amount of blood contained in them ; the result of this must necessarily be increased rapidity in the movement of the blood through the capillaries and veins which are in a state of inflammatory dilatation. But this, furthermore, results in increased secretion, and therefore even in this way diaphoresis acts antiphlogistically upon the inflamed kidneys. In addition to this, too, the profuse perspiration depletes the general systemic circulation, for the sweat is derived from the blood, and does not represent a direct transudation of the dropsical fluid. Now, although the vessels promptly reach their former state of repletion again by the absorption of this dropsical fluid, still it cannot be doubted that a certain period of time must elapse before this is effected. All this is time gained for the vessels of the inflamed kidneys—time for them to contract themselves to smaller calibres ; and this cannot be without its effect upon the function of these organs, clinical experience also proving that this effect is obtained. Patients urinate more freely just in proportion as the circulation, relieved from obstruction by dint of the diaphoresis, increases in speed, and the urine contains less albumen in proportion as the secreting vessels lose their state of preternatural distention. Finally, I hold it to be established that the disturbances of nutrition provoked by inflammation may be completely set right by a sufficiently prolonged and consistent diaphoretic treatment.

I am at present unable to decide what is the best and most efficient way of producing diaphoresis in chronic nephritis, since in my own Clinic I have not had the means of trying all the known methods. I have only operated with the so-called Russian steam-bath, and with ordinary bath-tubs, for the use of hot water, and of late years have only been able to avail myself of the last named, since our Russian bath has been disused from the bad state of repair it was in, and we have not yet had it replaced. I especially regret that I have been unable to try hot-air baths (the so-called Turkish), because I should expect them to act in an especially curative manner, inasmuch as this plan of applying the diaphoretic treatment undoubtedly offers the opportunity of producing the longest sweating without any prejudicial effects upon the patient, and without any special discomfort. The employment of hot steam and water baths unavoidably induces considerable elevation of the temperature of the whole body, if the bath is taken hot enough to cause profuse perspiration. This cannot be endured for more than a short time without injury. No similar overheating of the body need be induced in hot-air baths, and we should therefore be able to allow our patients to sweat for much longer periods without anxiety. The Russian steam-baths are certainly the most dangerous, both because in them it is most difficult to exercise the proper oversight over the patient whilst in the bath, and also by reason of the extreme height to which the temperature of the body may rise while in them. I would not, therefore, recommend them in the treatment of renal disease. According to our own experience, there has also appeared to be some difficulty in regulating the temperature of the steam at a uniform point, since the pressure under which the steam is driven into the bath-room is subject to no inconsiderable fluctuations.

This last objection does not hold good against the hot-water bath. If we wish to employ hot tub-baths for the above-named purpose, the water must be heated to a temperature of 40° Celsius (104° F.), and during the patient's stay in it this heat must be maintained by additions of hot water. To produce a proper sweating with a bath of this kind the patient must remain at least an hour in the water, and then lie sweating for several hours more wrapped in blankets in a bath-room heated up to the requisite pitch. If the patient becomes faint either in the bath or during the after-sweat, a glass of strong wine should be given him. Hitherto no experiments have been instituted to decide the question of how long a patient may stay in a hot bath without hurt, nor how often these may be repeated. And yet I would here call to mind the fact that Hebra kept his chronic pemphigus cases in a warm-water bath for one hundred days and nights continuously, and cured them in this way; and I am of the opinion that the diaphoretic treatment of chronic nephritis is capable of being considerably developed and perfected in this direction.

From a theoretic point of view, however, I should certainly prefer hot-air baths to hot water and steam, since in this way we surely avoid the overheating of the body, which is always considerable by the latter methods, and which must damage the strength of the patient. It would be at once simplest and most convenient to let these patients take their baths in complete establishments, with comfortable bath-rooms; but to meet special contingencies we could avail ourselves of makeshifts, so-called sweating-closets, or hot sand-baths. Indeed, these would enjoy a certain advantage over the regular Turkish bath, for the air which the patient breathed would be at a normal temperature, and the regulation of the temperature of his body would be easier to accomplish. Still, with this and similar makeshifts, the actual regulation of the temperature of the air of the closet, and still more that of the sand in a sand-bath, presents no slight difficulty. I have repeatedly had occasion to notice the consequences of lack of due caution in this respect.

I cannot omit here to refer to a piece of lay practice or village doctoring which prevails in some districts, and which proves that even the instinct of the common people has led them to employ the plan of diaphoresis for dropsy. I have repeatedly seen dropsical renal cases immersed up to the arm-pits in sacks of lentil meal, the skin of these patients being kept in a continuous perspiration from contact with the meal.

While, therefore, in my opinion, this diaphoretic treatment is the only one by which we can produce any effect upon the processes of inflammation taking place in the kidneys, without damaging the health of our patients, and while I regard it as at once the most effectual, and, in fact, almost the sole practicable method of cure that we can employ against that most troublesome symptom of the disease, the dropsy, I do not exclude the simultaneous employment of harmless, and especially of saline, diuretics. But I am bound to say that I cannot accord the same measure of praise to them as other writers do, either as a means of benefiting the dropsy or of restoring disturbed renal functions.

But other consequences and symptoms of this complaint make no less demand upon the attention of the physician who undertakes its treatment. Among them I refer especially to *disorders of the digestive organs* and to *anœmia.*

First, then, the diet must be regulated so as to suit the particular state of the organs of digestion. Care must be taken to provide the patient with the most abundant practicable compensation for the loss of albuminous matter to which he is subjected. This problem is one often rendered more difficult to solve by the dislike which the patients themselves so often evince for a meat diet. The use of hydrochloric acid and pepsin falls far short of meeting the indications in every instance in which the patient's own digestive apparatus fails to fulfil its task. Still less do stimulants, such as bitters and the like, remove the anorexia of these patients.

The experience that relieving the dropsy by exciting abundant sweating usually restores the appetite, appears to me most completely to support my view that the apepsia of chronic nephritis is to a great degree the result of œdema of the gastric and intestinal mucous membrane. So long as the patients manifest an invincible repugnance to meat, one may, as Niemeyer has proposed, try to attain a sufficiently abundant supply of albuminous material by a "*milk cure.*" But even this endeavor occasionally fails, since every patient is not in a state to take and digest large quantities of milk. Many patients tolerate freshly-made buttermilk much better than fresh milk. In our own district buttermilk is a favorite drink of the common people. Many of the renal cases in my Clinic are in the habit of taking large quantities of it daily, and to this circumstance I do not hesitate to attribute the good state of general nutrition that these patients evince in spite of the considerable loss of albumen they are daily experiencing. Furthermore, this large consumption, whether of milk or buttermilk, undoubtedly also has a beneficial *diuretic action.*

In most cases we are compelled to feel our way by experimenting, and thus discover the right method of treatment, both with regard to alimentation and medication. If, now, we succeed in removing the dropsy and the hydræmia which constitutes its immediate cause, the natural appetite and orderly digestion in most instances likewise return.

Whether any grave functional disorder of the digestive organs accompany the progress of chronic nephritis or no, the pallor of the mucous membrane will always inform us of the diminution of the red blood-cells produced by the albuminuria; and this is an indication for the employment of *ferruginous preparations.* I do not think that it matters specially what particular preparation is employed; in deciding this I am always guided by the individual case. When the patient can swallow a pill well, I prefer a mass which consists of sulphate of iron and carbonate of potash, in equal parts, to every other preparation, and so divide the pills that each one of them contains five centigrammes (about three-quarters of a grain) of the iron salt, directing that three or four of them should be taken after each meal. If the patient prefers the fluid form, I either give the syrup of the iodide of iron, a solution of iodide of iron in bitter tincture, or else choose some one of the ordinary tinctures of iron. But it appears to me of importance that these chalybeate preparations should, under all circumstances, be introduced into a full stomach. Beyond this, one need only endeavor to supply whatever preparation the patient best tolerates.

As to the therapeutic measures which may be necessitated in the course of the disease by the complications which may arise in it, as for example by phlegmonous inflammations, gangrene of the skin, pneumonia, pleurisy, pericarditis, peritonitis, etc., there is no need for me to say more here than that they must be directed in each particular case upon general principles. There is one remark, however, which I cannot omit, and that is that I do not favor the practice of making scarifications for the relief of the anasarca. The dropsy may be subdued by other means with more durable results. Cuts made into œdematous skin readily become starting-points for erysipelas and phlegmonous inflammations, and besides they are open to the objection of wetting the bed, thus causing all kinds of inconvenience and harm. I have mentioned above the punctures proposed by Boeck, to be made by the fine canula of a hypodermic syringe.[1]

The treatment of the uræmic attacks occurring in chronic nephritis should be conducted upon the same principles as those laid down by me in the previous chapter upon Acute Nephritis.

4.—Interstitial Inflammation or Induration of the Connective Tissue of the Kidneys. Genuine Contracting Kidney. Granular Atrophy of the Kidney. Renal Cirrhosis. Renal Sclerosis.

That pathological state of the kidneys at present designated by the above variety of names represents the third stage of what is known by authors as Bright's disease, and is alleged to be the final result of diffuse nephritis. It is supposed that the process is introduced by a stage of hyperæmic swelling, corresponding to acute parenchymatous nephritis; that this is followed by a stage of infiltration, our chronic parenchymatous nephritis; and that from this is finally developed the shrinking or contraction of the organ after the absorption of the infiltration.

Clinical observation, however, first convinced me that this traditional idea entertained in Germany does not represent the real truth of the matter. When I came attentively to watch and follow the symptoms which marked the whole course of the disease in those cases whose ultimate anatomical issue was the condition known as the Bright's disease of the authors, I found that no symptoms of acute parenchymatous nephritis opened the chapter of the disease, and that no signs of chronic parenchymatous nephritis appeared as precursors of the last but characteristic group of clinical features which accompany progressive contraction of the kidneys, features which Traube has painted with such masterly skill. Indeed, I came to the conclusion that this form of kidney atrophy was the result of a pathological process of its own, and one that began and followed its course quite independently of the process of diffuse renal inflammation previously described. I think I shall be able to adduce anatomical proof that this view, first suggested by clinical observation, is the correct one. At present, however, I must still leave the question undecided as to whether exceptional cases (compare that already adduced of Herr Kr., Case XVII.) may not occur where previous parenchymatous inflammation gives the impulse that determines the development of this peculiar process of contraction.

---

[1] *Reichert* and *Du Bois'* Archiv. 1873. S. 620.

I shall develop my views of the nature of this whole process later on. I will here only remark that so far as this is concerned, the opinions of writers, and especially those of English writers, who recognize this affection to be independent of parenchymatous nephritis, do not entirely agree.

Etiology.

The difference of opinion referred to above also makes itself apparent in the accounts given of the etiological conditions under the influence of which primary atrophy of the kidney is most frequently observed.

Gull and Sutton,[1] who regard this condition of the kidney as merely constituting one of the symptoms of a more or less widespread general affection of the entire arterial system, describe this last, as well as the renal change, as a disease of old age. They support their views by a table in which are collected the results of 336 post-mortem examinations, undertaken on the bodies of persons of different ages, selected without reference to the disease of which they died, and showing the manner in which the renal disease in question was distributed among the different periods of life of those examined. From the figures given, it appears that, among the 336 subjects that form the basis of this estimate, genuine contracting kidney occurred more frequently in proportion to the increasing age of the individuals. Thus, among 44 subjects who were between 10 and 20 years of age, atrophy of the kidney occurred but once, while among 13 who were between 60 and 70 years of age, atrophy occurred twelve times. It is not stated whence these two observers obtained the material for this examination, but every experienced physician, whose studies in pathological anatomy have not been confined exclusively to bodies obtained from hospitals and pest-houses, will admit that the proportions figured out by Messrs. Gull and Sutton have no claim to be regarded as of general applicability.

---

[1] On the pathology of the morbid state commonly called chronic Bright's disease with contracted kidney (arterio-capillary fibrosis). Medico-Chirurgical Transactions. Second Series. Vol. XXXVII.

At the same time I am perfectly ready to recognize the fact that genuine contracting kidney, or renal cirrhosis, is a rare occurrence in youth, but happens most frequently during mature or middle life. My own experience entirely contradicts Gull and Sutton's assertion that contracting kidney is a "disease of old age."

It is true enough that among my patients there were two old people of seventy years and upward, and no child as young as nine, as was reported in the work of these two English pathologists; but the small number of cases put forward in the following table of deaths, *verified by autopsies*, out of my clinical and private practice, are enough to show that, *influenced by love for their newly created conception of Bright's disease*, Gull and Sutton have evidently confounded the changes wrought in the kidney by old age with true and genuine contracting kidney.

Among the persons who have died, in my practice, of genuine contracting kidney, *and whose bodies were examined after death*, the entire number being thirty-three, it proved that

1 was 18 years of age.
1 was 19 " "
2 were 20 " "
4 between 20 and 30
9 " 30 " 40
4 " 40 " 50
7 " 50 " 60
5 " 60 " 73

From this table I have excluded every case in which no post-mortem was made, to avoid all error which a mistake in diagnosis might introduce.

It is true that the following table from Dickinson, representing 308 cases of death by granular degeneration of the kidneys, tabulated according to age, shows results which differ considerably from my own; still, it offers no proof at all of this affection being a special disease of old age. Among the cases here collected, there is not a single one under ten years of age.

[1] Between	11 and	20	years,	1	case.
"	21 "	30	"	24	cases.
"	31 "	40	"	50	"
"	41 "	50	"	93	"
"	51 "	60	"	76	"
"	61 "	70	"	47	"
Over 70 years,				17	"

Still more remarkable than this difference in the frequency of the occurrence of genuine contracting kidney at different periods of life, is its relatively different frequency *in the two sexes.* Thus, according to English writers, it is twice as common among men as it is among women. Among the thirty-three cases tabulated above by me, seven only implicated women, a ratio, therefore, of more than 4 : 1. The cause of this may, perhaps, be that our hospitals are generally more frequented by men than by women.

---

[1] I have taken this table from Lecorché's work, "Traité des maladies des reins." I was unfortunately not able to procure Dickinson's work, "On the Pathology and Treatment of Albuminuria," as it is long since out of print.

The disease occurs in *every class of society*. I have observed it in country people, field-laborers, servant-maids, mechanics employed in the most various branches, tailors, seamstresses, merchants, officials, literary men, artists, etc., and my experience hitherto does not enable me to accuse any particular calling or occupation of predisposing to this affection. I should especially contradict the results of my own observations if I were to admit that out-door occupations, or such as proffered frequent opportunities of exposure to wet and cold, rendered persons more prone to this disease than regular work in the study or in the counting-house.

Independently of avocation, I am acquainted with no particular circumstances or habits of life which can be confidently designated as being the cause of this disease. Above all, I must enter my protest against the view which is widespread in England, although Dickinson certainly disputes its correctness, that the abuse of spirituous liquors favors the development of the genuine contracting kidney. In the first place, among all the patients whom I have treated, three only were brandy drinkers to any notorious excess, while the greater number by far who were affected with this complaint had lived remarkably abstemious lives. In the second place, throughout my twenty-five years of active service as a hospital physician, I have had the most abundant opportunity of watching the consequences of intemperance both at the bedside and upon the post-mortem table; yet these three cases have hitherto been the only ones in which I have found atrophied kidneys in the bodies of habitual drunkards. That British experience should be different may, perhaps, arise from the fact that alcohol is taken in more concentrated form there than it is in this country, and is more frequently mixed with juniper oil (gin, as it is called). It appears self-evident that substances experimentally found to stimulate the activity of the kidneys must, if taken in great excess, induce pathological changes in these organs. The following two cases, perhaps, also admit of the same interpretation.

Case XVIII.—On the 25th September, 1858, a musician of forty-eight years of age, who was living in easy circumstances, was attacked with erysipelas of the face and scalp. He was brought to my wards in a state of complete insensibility, and two days later died. Up to the date of his erysipelas he had reckoned himself quite well, and was deemed healthy by his family and friends, for he had carried on his occupation as a teacher of music without any interruption.

Yet trustworthy information reached me that it had for many years been his habit to consume not less than twenty pints a day of the ordinary strong beer that is sold here. We found his kidneys contracted in an extreme degree, and there was considerable hypertrophy of the left ventricle, but no trace of dropsy or of older organic disease.

Case XIX.—The second case occurred in a merchant here, Mr. L., who was fifty years of age. This gentleman had apparently enjoyed uninterrupted good health, in spite of a life passed in a continual vortex of exceedingly stirring events, as an ardent patriot and venturesome business man, and notwithstanding the almost incredible exertions with which he had taxed himself by forced journeys and by night work in his office. There was one thing only which often troubled him—his tendency to obesity. Neither his repeated and excessive emotional disturbances, nor the fatigues and labors to which, as merchant and political agitator, he subjected himself, had ever led him into any excess in taking wine or alcohol in any form. But, on the other hand, it had been his habit for many years, both when travelling and at his desk, to drink enormous quantities of strong tea, sometimes as much as ten large cupfuls a night. It happened that I often met him in society, and I was struck by the comical straits to which he was often reduced, when in company, by his frequent desire to make water. The small capacity of his bladder was the subject of a good deal of banter.

In 1869 he felt unwell the entire summer, repeated attacks of vertigo and palpitation being the commencement of his difficulties. Soon he had attacks of asthma. But it was not till the beginning of December that his ankles began to swell. This induced him, for the first time, to submit to a medical examination, when the albuminuria and great hypertrophy of the heart, associated, however, with all the symptoms of cardiac debility, were discovered.

On the 5th of February he died. The post-mortem, which was, unfortunately, not undertaken until after the body was much decomposed, showed a large amount of general dropsy, contracted kidneys, amyloid degeneration of the spleen; considerable hypertrophy of the left ventricle, but extreme friability and softness of the heart's muscular tissue. I narrate this case merely as a matter of fact, but as no other similar observation is known to me, I do not venture to attribute the renal disease in this case to the abuse of tea.

Since Olivier[1] first directed attention to the occurrence of albuminuria among lead-workers, and expressed his opinion that chronic lead-poisoning might lead to degeneration of the kidneys, the view has been pretty generally entertained in England that lead-poisoning is a cause of granular atrophy of the kidneys. Grainger Stewart found this malady several times among those who worked with lead, and in connection with other symptoms of chronic lead-poisoning. Out of forty-two cases of chronic lead-poisoning, Dickinson records having seen twenty-six die of renal atrophy. Among German writers, Rosenstein is the only one who, to my knowledge, has considered the connection between lead-poisoning and renal disease, and he, fortified in his conclusions by experiments made on dogs, completely denies their interdependence. One of my patients had had severe lead paralysis, the result of his having been poisoned by snuff that contained lead, and which he was in the habit of taking to an enormous extent, but this occurred years before his renal disease was discovered. He died of cerebral apoplexy, and I never saw so large an effusion of blood in the cavity of the cranium as was found in this case. I merely mention this to show that the statement that apoplectic effusions in renal atrophy are always inconsiderable is by no means invariably true.

---

[1] Gazette Hebdomadaire. 1863. Nos. 10 and 27.

It appears to be an undoubted fact that gout—hence, presumably, the contamination of the blood with uric acid, or rather with the salts of this acid—may give occasion to genuine contraction of the kidney. All English writers, at least, are united on this point; indeed, in England the disease often bears the name of "gouty kidney." Rayer also distinguishes a "néphrite gouteuse." In Germany, where gout is so much less common than in England, much less attention has been given to the relation of this kidney disease to gout. Among my own patients there was only one gouty subject, a high official of fifty-two years of age. But I have certainly had small opportunities of studying true gout, and am therefore from my own experience in no position to judge of the frequency of renal disease in gouty subjects.

Lastly, I must mention that several of my patients had had inveterate gonorrhœa, with extension of the gonorrheal catarrh to the bladder. In two of them I made out the renal disease while the bladder trouble still existed. The question hence suggested itself to me whether gonorrhœal inflammation of the mucous membrane of the urinary passages extending up to the pelvis of the kidneys might not have entailed the subsequent renal affection. Liebermeister raises the same question in detailing a case of renal contraction in his work entitled "Zur Pathologischen Anatomie und Klinik der Leberkrankheiten," p. 75.

I know of nothing further to be said with regard to the etiology of genuine contraction of the kidney. In the great majority of my cases I have been obliged to renounce the attempt to fix upon any circumstance which might be assigned as the cause or origin of the disease.

Summary of the Progress of the Disease.

Genuine contracting kidney may have existed for a considerable time, and may have reached a serious degree of development before the patient becomes aware, by any kind of symptom, that his health is no longer as good as it used to be. This fact is attested by the no small number of cases in which the kidneys have been found in a state of advanced atrophy or contraction in persons who have died of some intercurrent disease, and who, up to the date of the occurrence of the latter, have carried on their usual avocations without let or hinderance. An instance in point may be found in the case of my musician, mentioned above.

It still oftener happens that patients die either suddenly of the immediate results of their renal malady, or after a short illness of such indistinct kind and trifling nature that they disdain to summon medical advice. Thus, I have repeatedly witnessed an epileptic attack as the first as well as the last symptom of this disease that has attracted attention, the seizures being repeated one after the other, and occasioning death within a very short period; and then after death I have discovered extensively contracted kidneys as the real cause thereof. This has happened with persons who up to the date of the catastrophe had carried on their work without interruption, and had both fancied themselves and been regarded by their friends as perfectly healthy.

Case XX.—On the 3d of March, 1859, I was summoned at ten o'clock at night to the gardener in our Botanic Garden here, B., aged twenty-seven years. He was a large, stoutly built, muscular man in capital nutrition. His mother was insane, and he himself at sixteen had exhibited some symptoms of melancholia for a short time, and at twenty had for a period of three weeks' time suffered with repeated attacks of epilepsy. Since then he had been perfectly well. A few days before his present attack he noticed a peculiar twitching of his underlip, and the same thing struck the attention of his sister, who kept house for him. On the morning of the 3d of March he felt uncomfortable, but without knowing how to explain his being so; was rather lachrymose, and at his dinner ate less than he was wont to. Still his feeling of not being well did not prevent his going out to a reading entertainment, and taking his part in the reading of a drama in character. He read his part without hesitation, but was suddenly interrupted in the middle of his reading by an attack of general convulsions, followed by complete insensibility. I found him still in the assembly room, which was filled with tobacco smoke, and when he came to himself again I had him removed to his home. He complained of sickness and slight giddiness, looked very pale, and felt very weak, but he had no headache. His pupils were moderately dilated, reacted well to the stimulus of light. Pulse 92, soft and full. During the night he had two more attacks of convulsions, followed by sopor of short duration. On the morning of the 4th of March he was quite himself, but felt excessively poorly; he had no appetite, but was in no pain; had passed no urine. Pulse 84. Ordered bleeding and Epsom salts, cold applications to the head. In the afternoon he had another fit; in the evening he was rational, and complained of headache; still he had passed no water. The bleeding was repeated. Immediately after my visit at eight o'clock in the evening he had another attack of convulsions, and these were so often renewed during the night that the patient between whiles never recovered his senses. Fæces and water were passed under him.

On the morning of the 5th of March, I found him in profound coma. Pupils small and fixed, not reacting to light; a profuse clammy sweat bedewed his whole body. By means of a catheter a few drachms of urine were drawn off from the bladder; the urine was thick, contained a good deal of albumen and numerous casts, among which were several of a dark, granular form. That afternoon he recovered sensibility once more for a short time. But then ensued a series of fits, which, with short intervals of profoundest coma, continued up to the morning of the 6th of March, when he died.

The fits commenced with great regularity by twitching of the muscles on the left side of the face, and then attacked the left arm and afterwards the right arm, extending next to the lower extremities, and finally the muscles of the trunk were convulsed in the most terrible manner. These attacks sometimes lasted over two minutes and were succeeded by snoring, stertorous breathing.

I append the abstract of the post-mortem examination: Enormous hyperæmia both of the membranes of the brain and of the brain itself. Brain substance otherwise normal. Subpleural ecchymoses. No serum either in pleural cavities or pericardium. Interstitial emphysema upon anterior surface of both lungs—upper lobes. The alveolar structures of the upper lobes of both lungs were blown up with emphysema, and were of a rosy red color; the posterior portions of the lungs were of a dark black-red color from excessive hyperæmia, and were œdematous, but still contained air. Considerable hypertrophy of the left ventricle. Heart firmly contracted and peculiarly shaped at its apex, for the right ventricle was not enlarged. Valves normal. Heart's weight 360 grammes. Hemorrhagic infiltrations streaked the mucous membrane of the stomach and the lower portion of the ileum. Both kidneys were noticeably diminished in size. Capsules firmly adherent, thickened, small portions of the renal structures adhering to them when torn off. The surface of the organs was granular and covered with perfectly uniform nodulations. Color dark brown. Consistence firm and tough.

On section, it was seen that the diminution in size involved the cortical substance principally, the pyramidal portions much less. The entire parenchyma was hyperæmic. A small quantity of thick urine was found in the bladder. Unfortunately, no minuter microscopic examination of the kidneys was instituted, and all I find in my journal is the notice that some of the uriniferous tubes were considerably bulged and dilated, and filled with finely granular detritus and minutest fat particles.

In other instances an attack of *cerebral apoplexy* terminates life with equal suddenness, and also frequently without any warning, while the individual is engaged in his business or in the midst of society.

Thus, one day a robustly built master carpenter was suddenly struck down insensible in a new house where he was engaged at work, and brought thence to me at the hospital. He died immediately after his admission. We found apoplectic effusion of blood into the right hemisphere of the brain, which had broken through into the right ventricle, as well as hypertrophy of the left chamber of the heart and contracted kidneys.

But in the larger proportion of cases striking symptoms indicative of the disease precede its fatal termination, and are prolonged sometimes over a period of several years; and in every instance where the renal malady reaches its extremest grade, and so to speak, exhausts itself, not having been interrupted by any one of the above-mentioned accidents, a really long illness is experienced before the end comes.

Under any circumstances it may be considered as established that the special process of contraction in the kidneys, which we are discussing, must have made considerable progress, and therefore have lasted for some considerable time, if we are able to diagnose it with certainty at the bedside. It is probable that its actual first beginning always eludes diagnosis, and furthermore the symptoms which accompany the development and progress of the affection, at least in its earlier stages, are not calculated to direct the patient's own attention to his kidneys as the part at fault. The only thing which disturbs these patients is the frequent desire to pass water, which is principally apparent at night. In a previous publication I have already called attention to the fact that the patients themselves generally consider this troublesome symptom as a bad habit, and attribute their other sensations of malaise to the effect of taking cold, which they suppose they must have been exposed to in getting up to make water during the night. Neither pain nor any other discomfort in the renal region appears to attend the affection at any period of its course.

The other initial manifestations of the disease vary greatly in different cases. At one time they proceed from the heart, and the patients complain of occasional *attacks of palpitation* accompanied sometimes with *vertigo;* more often they occur with a sense of *great uneasiness*, or with the feeling of *suffocation* or *want of breath.*[1] Then, upon carefully examining the chest, one finds that *the heart is enlarged.* The area of precordial dullness extends upwards and to the left beyond the normal limits. The apex-beat may be in the normal situation or a little farther outward to the left; the action is increased and occasionally heaving. With the stethoscope one finds that the heart's sounds are very loud, and there is an especially flapping character attached to the accentuated diastolic sound at the origin of the aorta, heard loudest to the right of the sternum, just above the point of insertion of the third right costal cartilage.

Any hypertrophy of the left ventricle, without any valvular lesion of the heart to explain its occurrence, ought to direct attention to this renal malady as possibly existing.

Corresponding to this hypertrophy of the left ventricle we find the radial pulse remarkably tense and bounding. Traube says, rightly enough, that one can recognize the renal disease by the pulse.

---

[1] *Lecorché* says that the palpitation, at night time especially, amounts to actual torture with many patients. I have never seen such extreme inconvenience as this from it in any of my renal cases.

But these symptoms belonging to the circulatory apparatus do not always continue to the end as I have just described them. It is true that the hypertrophy of the left ventricle is never lost, but in the further progress of the disease the general nutrition suffers from the disorders of digestion (which we shall shortly mention), and this malnutrition makes itself apparent, too, in the hypertrophied heart-muscle, whose fibres become the seat of fatty degeneration. The impulse of the heart grows feebler, and is often hardly perceptible towards the end of life; the heart-sounds, previously so loud, become faint and indistinct, and the radial pulse gives merely a feeble, easily compressible pulse-wave.

Here I must distinctly state that I have never failed to observe the objective signs of hypertrophy of the left ventricle in any of my cases of genuine contracting kidney, or to confirm the fact in every post-mortem made upon their bodies, and therefore can never hold that a diagnosis of this renal disease is made certain when no enlargement of the left ventricle is recognizable. Contrary, however, to the statement of M. Lecorché, I must add that I have only exceptionally heard my patients complain of the annoyance they themselves derived from palpitation; far more often they were entirely free from any such trouble. Lastly, I will say that other writers have failed to observe the hypertrophy of the heart in some cases of far advanced renal contraction.

Apart from the difficulty of breathing incident to the attacks of palpitation, disorders of the *respiratory organs* but rarely occur during the early period of this disease. It is true that attacks of cold and bronchial catarrh in some cases exhibit extreme obstinacy before the renal affection is recognized, and occasionally induce a rapidly fatal œdema of the lung even in cases which are but little advanced. I saw an instance of this once in a butcher's apprentice of twenty years of age, who the day before his death was engaged at his craft, and whose body, after death, did not show a trace of dropsical effusion elsewhere. Much more troublesome are those attacks of bronchial catarrh that are incident to the later stages of the complaint, and occur when the patient is already reduced in his general nutrition. To be sure, the bronchial secretion in these cases is ordinarily scanty; but a specially inveterate and incessant inclination to cough frightens the patients, and leads many of them to think that they are consumptive—an idea in which they are confirmed by the extreme dyspnœa which assails them, whenever they make any slight exertion, and which is due to the palpitation of the heart and the anæmia.

In the very advanced stages of the disease, too, the patients very often have fits of dyspnœa exactly like nervous asthma. Like this, too, the seizures happen principally at night, and pass off entirely during the day, but they are mostly severe enough to compel the patient to sit upright in bed. On examining the lungs they are found to be full of air; and during the attack one hears, with the stethoscope, sibilant and sonorous sounds widely diffused throughout both lungs, but, just as in nervous asthma, principally during expiration. During the asthma fit the patients occasionally are manifestly cyanotic. Very often these attacks end, like those of nervous asthma, with short, repeated coughs and expectoration of a more or less abundant frothy fluid. At first, during the intervals between the attacks, one hears perfectly normal vesicular breathing sounds throughout the lungs. Soon, however, the attacks increase in frequency, but appear to be less severe, while they leave some permanent though moderate degree of dyspnœa during the intervals; the cough and expectoration of watery fluid mixed with scanty pellets of mucus become more obstinate. The stethoscope then reveals fine moist crepitant râles throughout both lungs. Some of these patients finally die in an attack of œdema of the lungs.

In other cases, the first evidence of the disease consists in frequent and severe *attacks of headache*, sometimes occurring under the form of excessively severe *hemicrania* of remarkably long duration; indeed, I have observed this form a number of times. The pain, too, may extend down the neck, and even to the brachial plexus of the affected side. Such paroxysms may last for several days. In one case of this kind I noticed almost complete anæsthesia of the skin of the face and of the fingers upon the affected side during the continuance of the attack. This patient had an attack of the kind nearly every week; and finally, during the intermissions, normal sensation did not quite return to the face or fingers, but a feeling of numbness and formication remained.

In any case of migraine occurring after middle life in an apparently robust individual, it is well to consider the possibility of the nerve affection being dependent upon contracting kidneys. Neuralgic pains in the track of other nerves have, in my own experience, proved far less common than headache, so that I am still doubtful whether I can properly connect these isolated attacks of neuralgia with this renal disease as cause and effect. But the terrible itching of the skin, which I shall advert to hereafter, belongs indisputably among the results of the renal affection, and it is likely enough that a more or less extensive muscular pain that sets in towards the end of life, and is usually satisfactorily but fancifully explained under the name of rheumatism, owns a similar origin.

Sleeplessness or unrest, the sleep being broken by troubled dreams, may be reckoned in some cases among the earlier symptoms.

Very often some *disorder of vision*, provoked by the specific structural change that has taken place in the retina, forms the first event that attracts the patient's own attention. No small number of my patients first went, on account of their eyes, to my colleague Voelckers, our esteemed ophthalmic surgeon, to whom I am indebted for the supplement to this work upon Retinitis albuminurica, and were first induced by him to place themselves under medical treatment, which they were not aware they needed. In this early stage patients rarely complain of any disorder of digestion or of a capricious appetite, although nausea and inclination to vomit are more common. Occasionally I have noticed a most repulsive smell in the breath of my patients, even at a very early stage of the disease.

The rule certainly is that for years the appetite and digestion are absolutely unaffected in this complaint, and the general nutrition therefore continues perfect. For this reason the patients preserve their strength and power of getting about, go to their employments without fail, and do not excite the slightest suspicion, either among their neighbors or in their doctor, of being the victims of an insidious malady which is steadily undermining their health and surely bringing them to a premature end.

When the disease, however, has reached a certain stage, dyspeptic disturbances almost invariably present themselves, and during the later periods of the complaint these are perhaps the most constant of the secondary symptoms. The *appetite* fails more and more, and after meals the patient is racked with *dyspeptic pains;* repugnance to a meat diet is especially apparent, while sickness and often vomiting occur after eating, events which make the patient more and more sparing in the amount of solid food that he takes. In many patients, on the other hand, we find a most troublesome *thirst*, which compels them to take large quantities of fluid; and since they pass a good deal of urine in consequence, intelligent individuals, by noticing this symptom, are quite often led to suppose that they are suffering with diabetes mellitus. Indeed, this was the circumstance which led Mr. L., the merchant, whose case I related on a preceding page, to consult me.

In consequence of dyspepsia and frequent vomiting, there next ensue *emaciation*, *anæmia*, and *loss of strength.* The skin acquires a peculiar dryness and becomes blanched or, not infrequently, assumes a dirty faded color. It is then, when nutrition has become disordered and the strength is gone, that the patient first realizes that all is not well with him. As a rule, too, at the same time and in consequence of his malaise, a peculiar gloominess and moodiness of spirits come over him. These patients are far more irritable than is their natural wont. Several of my male patients have complained to me quite early in the course of their disease of the *diminution* or *loss of sexual power.* In two of them I found seminal filaments in the urine sediment.

Most patients *do not exhibit* any *dropsy* throughout the entire long course of genuine renal contraction. Before the kidney tissue is so completely dwindled away that what remains of it will no longer suffice to remove the excess of water from the blood, the larger number of the patients die either of *uræmia* or *apoplexy*, or of *inflammatory exudations into the serous cavities*, or of *inflammatory infiltration of the lung tissue*, or else of *erysipelatous* and *phlegmonous inflammations of the general surface* of some part of the body.

If, however, in the progress of the disease some disturbance of the circulation sets in, as some defect in the valves of the heart caused by endocarditis,[1] or a pleuritic effusion occurs, or some acute exacerbation of the existing bronchial catarrh arises, or if a notable weakening of the vigor of the hypertrophied heart's muscle takes place through fever or other complication, then œdema may occur early before the process of contraction in the kidneys has made any serious advance. Ordinarily, the œdema is limited to the ankles, and if it has been apparent by day when the patient was about, still it subsides by night when he is in bed. More rarely the anasarca first of all invades the face and eyelids of these patients when they are feverish and confined to bed. It very rarely happens that this anasarca, which is provoked by some intercurrent affection of short duration at an early stage of the complaint, is very extensive. It subsides forthwith upon the removal of the determining cause, and this usually without returning again.

It has only been in cases where endocarditis with mitral insufficiency coincided with a copious effusion into the right pleura, and was attended by fatty degeneration of the hypertrophied heart's muscle, that I remember seeing a considerable amount of general dropsy lasting with pertinacity up to death,—the autopsy then showing, however, that the kidneys were only moderately contracted.

But when the disease advances to its extremest grade, so that there is nothing but the smallest remnant of kidney left capable of performing its functions, then dropsy will certainly exist. But, in these cases, anæmia, general wasting and loss of strength, precede the dropsy ; and contemporaneously with the occurrence of the ultimate anasarca there are other evidences of the insufficiency of the kidneys for its other functions, namely, the removal from the body of its nitrogenous waste, evidences which take the form of chronic uræmia, which will shortly terminate existence. For the reason last mentioned, dropsical swellings during the terminal stage of the process of contraction of the kidney seldom attain any great extent. In the larger proportion of cases the dropsy is confined to slight œdema of the extremities, of the male genital organs, or of the face—œdema which comes and goes, or changes its locality in accordance with the position of the patient. Thus, I have repeatedly seen the œdema entirely confined to the prepuce of the penis, and lasting a considerable time in this situation.

---

[1] I must prominently point out that endocarditis upon the valves is by no means so rare a complication of the hypertrophy of the left heart following contracted kidney. In two of my own cases the symptoms of endocarditis developed themselves in the course of the renal affection and under my own observation, leading, as confirmed by the autopsies, in the one case to mitral insufficiency, and in the other to the formation of considerable vegetations upon the ventricular surface of the semilunar valves of the aorta.

Dropsy less frequently terminates life, towards the end of the contracting kidney disease, by actual œdema of the lung, or flooding of the lung tissue with dropsical effusion. Still, I have seen not a few examples of lung œdema of a temporary nature, and noticed how distinctly this sometimes alternated with the general condition of anasarca of the surface of the body. These attacks of lung œdema were characterized by the sudden onset of extreme dyspnœa, advancing to absolute orthopnœa, and attended by a bloated cyanotic condition of the face and pertinacious cough, whereby considerable quantities of frothy watery sputum were frequently expectorated. Physical examination of the chest showed that less air than normal was contained in the lungs, and that this was principally apparent in the posterior, though sometimes also in the anterior, portions of the organ, the parts thus affected giving a somewhat muffled and tympanitic note on percussion. The stethoscope, furthermore, revealed the familiar fine crepitant râles over the areas corresponding to the altered resonance note. Both things—the altered percussion note and the characteristic fine crepitant râles—may be confined to very limited portions of the lungs. Although these symptoms are, as a rule, merely transitory, disappearing after a short time, still occasionally they persist till death, and undoubtedly accelerate the end.

Nearly all the patients whom I have seen die in the extreme stage of atrophied kidney sank under symptoms of *chronic uræmia.* Dyspeptic troubles, and especially *obstinate vomiting*, usually open the train of these symptoms. The characteristic feature of this uræmic vomiting lies in the fact that it happens when the stomach is empty; in the morning, for example, directly upon awaking, and before breakfast. The vomited matters present a different appearance, of course, according to circumstances. Early in the morning, and before any food has been taken, the vomit is often nothing but a watery fluid of excessively low specific gravity, mixed with shreds of mucus, and the quantity then is seldom great. This fluid usually has a feebly acid reaction. I have never been able to discover urea in it. In two cases it certainly presented a strongly alkaline reaction (in the one it was always mixed with blood) and gave off a pungent smell of ammonia. In both examples the clear filtrate, collected and treated with acetic acid up to distinct acidulation, developed abundant bubbles of gas. In some patients this uræmic vomiting only ensues after the ingestion of food or drink; after the vomiting, however, the desire for food still remains. Whatever these patients eat causes vomiting, and there seems to be no way of preventing it.

Diarrhœa, though a far rarer event in a case of contracting kidney than vomiting, may set in near the end and is apt to be as obstinate and irrepressible as the vomiting itself. The evacuations may be both excessively abundant and frequent, and the more frequently they are repeated the more fluid they become. I have always found the stools still colored with bile, but excessively fetid. I have not analyzed them more closely; but they might well have contained ammonia, though this would be of no consequence, for ammonia occurs in the alvine dejections under a variety of circumstances. In one case of the kind I found after death that the mucous membrane of the small intestine had been ulcerated in large patches, and that the calibre of the gut, in consequence, was in one place so contracted by cicatrization that dilatation of the portion of intestine immediately above had taken place, so as to form a sausage-like tumor, which could both be seen and felt, during life, quite distinctly, upon the right side over Poupart's ligament (vide Case XXI.).

The patient's strength is very rapidly reduced when either obstinate uræmic vomiting or diarrhœa sets in. If previously able to be up, they now take to their beds, and a constantly *increasing apathy* appears to overcome them. They become indifferent to what takes place about them, and sleepy, and then gradually subside into a somnolent condition, out of which it is at first easy to awake them. Soon, however, the senses become more dulled, until profound coma, which often lasts some time, ushers in death.

In some instances symptoms of irritation precede this gradual extinguishing of the nerve energies, and among these evidences of irritation I shall notice first excessive *itching of the skin*, which is a terrible annoyance, and one which occasionally robs the patient of his rest at night for months before the end, driving him, even when he lies half-insensible, to tear his skin with scratching it.

In other cases, for days before the final coma sets in, one observes *twitchings in certain groups of muscles*, or notices that the whole body is convulsed by the contraction of its muscles, in a jerking manner, just as if it had received an electric shock. It more rarely happens in these cases of extreme renal contraction that *general convulsions of an epileptiform nature* introduce the fatal coma, or even interrupt it when it has once set in. In one case of most advanced degeneration of the kidneys, I remember seeing epileptiform convulsions constantly recurring for three whole days, being followed by maniacal delirium, and succeeded again by coma of short duration, finally to end in a return to perfect sensibility. Death ensued here at last, with symptoms of collapse, just eighteen days after the last attack of convulsions.

Case XXI.—H. Hoyer, eighteen years of age, had in her childhood (*i. e.*, when she was under ten years old) had ague, measles, and scarlet fever, but she never was dropsical. Since that age she had experienced no illness, but at fifteen had some breaking-out on her face. At the commencement of the summer of 1872 she noticed something wrong with her eyesight, and for this reason presented herself at our ophthalmic clinic and had her eyes examined. Prof. Voelckers found she had retinitis Brightica, and, upon further investigation, discovered albumen in her urine and all the physical signs of a hypertrophied left ventricle, as well as a pulse of high tension. At the date of her examination the patient experienced no subjective sensations of ill-health or discomfort. In the course of the summer the affection of her eyes got worse, symptoms of dyspepsia arose, with frequent vomiting, and she complained a good deal of headache. Her mother had already noticed for years that her daughter passed a great deal of water.

On November 18th the patient's condition became quite suddenly worse. She had maddening headache and obstinate vomiting, so that no food could be taken at all. Her eyes were worse, and the urinary secretion was strikingly diminished. On the morning of the 20th she became completely blind. Repeated attacks of general convulsions occurred, to such extent that she was thrown out of bed. At 10 A.M. she was brought into the hospital.

State on admission: half comatose; slow, sighing respiration; frequent yawning; all questions answered by her saying, "I don't know," in a tired, reluctant tone. From time to time she made retching movements. General state of nutrition good; skin quite pale; slight œdema of the face and the backs of both hands; numerous petechiæ on the abdomen and lower extremities; great bruises over the right trochanter and over the right anterior superior spine of the pelvis. Another considerable ecchymosis was seen beneath the conjunctiva of the left eye; pupils large and scarcely acting. With the ophthalmoscope we could make out extensive and glaring white patches in both retinas. The tongue was bitten and swollen. Temperature somewhat raised. Pulse small, 90. Area of præcordial dullness increased upwards; sounds clear, but faint; the diastolic sound over aorta only was distinctly accentuated. No trouble in the lungs; nothing abnormal discovered in the abdomen on her admission.

In the course of the same day she had five convulsions of distinct epileptiform character, followed each time by deep coma, out of which the patient could not be completely roused between times. In the afternoon 200 c.c. of urine were saved (that previously passed having been in the bed); this was clear, sp. gr. 1021, and contained a good deal of albumen and a few small, pale casts filled with epithelium. Towards evening the patient took a little wine. 350 c.c. of blood were abstracted by venesection.[1] At night there was another attack of convulsions, followed by maniacal excitement. On the 21st of September the patient was more rational, but vomited in the evening; still, she was able to take some fluid nutriment.

Once during the night between the 22d and 23d of September, slight twitchings came on, without loss of sensibility; but this was the last time. On the 25th, the bowels up to this date having been shut up, and the patient having vomited some very sour stuff several times, diarrhœa set in, and never left off again. Each day as much as 1,200 c.c. of fluid materials, having for the most part an acid reaction, were evacuated by the bowels, while the flow of urine, which up to this date had been pretty copious, was diminished correspondingly (*vide* table *infra*). No urea could be discovered either in the fluid fæces or in the matters previously vomited.

---

[1] The blood examined for ammonia by *Kuehne's* method gave negative results. The alcoholic extract, however, on the addition of a concentrated solution of nitric acid, threw down a thick deposit of crystals like nitrate of ammonia. The specific gravity of the serum, estimated by the pyknometer = 1022.8. One hundred parts of serum contained 91.16 water and 8.84 solids.

Towards the end of the month a slight febrile movement set in, with some painful sensations in the abdomen, and upon examination I found a sausage-like tumor upon the right side, over Poupart's ligament. The abdomen now grew tympanitic, the diarrhœa more copious; her strength failed rapidly; there was slight anasarca of the ankles; and the patient died on October 10th, after being very deaf in both ears for the last few days.

Urine Table.

Date.	24 hours' quantity. c.c.	Specific gravity.	Urea.		Albumen.		Remarks.
			%	In toto.	%	In toto.	
Sept. 22....	525	1023	2.8	14.7	2.364	12.311	Reaction of urine always acid. Sediment: uric acid crystals and small hyaline casts in scanty numbers. At first white and red blood-cells; afterwards none.
" 23....	600	1022	2.4	14.4	1.528	9.168	
" 24....	1,200	1012	1.3	15.6	0.564	6.768	
" 25....	1,500	1013	1.2	18.0	0.592	8.880	
" 26....	1,100	1013	1.8	19.8	0.724	7.964	
" 27....	1,700	1009	1.3	22 1	0.210	3.570	
" 28....	1,200	1015	1.4	16.8	0.382	4.402	

From the 28th of October the entire urine could no longer be collected on account of the diarrhœa; still there was no doubt that the daily quantity diminished considerably, the specific gravity not rising. On October 4th, 700 c.c. were saved, and it was presumed that none had been lost; the specific gravity of this was 1012.

Post-mortem.—Both kidneys were atrophied; hypertrophy of left ventricle of the heart; purulent peritonitis; ulceration and stricture of the lower end of the ileum.

A medium-sized, somewhat emaciated woman; moderate anasarca of back of left foot; petechiæ on both thighs; abdomen moderately distended.

Skull cap thin, but very strong; membranes remarkably pale; brain substance also very pale, but firm; nothing else found.

Pleural cavities empty; nothing wrong in the lungs; heart firmly contracted: wall of left ventricle three centimetres thick; valves normal.

There was a considerable quantity of purulent exudation in the abdominal cavity. The great omentum was infiltrated with pus, and everywhere slightly stuck to the intestinal coils which it covered; in the right lower abdominal region it was firmly adherent to the intestine. Upon endeavoring to break down this adhesion the bowel tore, and allowed its slate-colored, gray contents to escape. The portion of intestine thus implicated was the lower end of the ileum, and it was also further attached to the fundus of the bladder and the anterior pelvic wall. The serous aspect of the entire small intestine was covered with purulent material, and its mucous surface even quite high up was strongly stained in patches by deposits of pigment. Towards the lower end of the small intestine were an immense number of irregularly shaped spots marking losses of substance where the mucous membrane had evidently been exfoliated.

Thus, for instance, close to the ulcers we found similarly irregularly shaped exfoliations of mucous membrane some centimetres long, and lastly string-like scars five centimetres above the ileo-cæcal valve, corresponding to the line of insertion of the mesentery. It was by this cicatricial formation that the calibre of the gut had been so narrowed, for a portion of about a finger's length, that it would hardly admit one's little finger. Fifteen centimetres above the ileo-cæcal valve, and beneath such an exfoliation of the mucous membrane, the intestine was perforated. There was nothing abnormal in the last five centimetres of the ileum or upon the ileo-cæcal valve.

The mucous membrane of the stomach was pigmented in spots towards the pylorus. *Liver normal. Spleen* of normal size; substance soft. Nothing wrong was found about the suprarenal capsules. *Left kidney* like a child's kidney. Capsule stripped off easily, small portions of the substance of the organ only being attached to it in a few places. Length 9½ cms., breadth 4½ cms. (3¾ by 1¾ inches); surface marbled, moderately smooth, but still in parts uneven from numberless shallow depressions; cortical substance much atrophied, especially so in the lower half of the kidney, where in places it measured scarcely three millimetres in breadth. The kidney itself was flaccid, but its substance tough; color of the cortex, pale yellowish gray; that of the pyramids pale flesh color. *Right kidney*, 8 cms. long, 4½ broad (3.1 by 1.7 inches), rather thicker than the left, but otherwise like it. The renal pelves of both organs were remarkably small; the mucous membrane of the same pale, tender, and anæmic. Both renal arteries were large.

At the commencement of these symptoms of nervous irritation, the result of uræmia, the patients are habitually in a depressed, morose condition, and when they are anyways equal to it in strength, ready to break out in fits of passion; maniacal attacks, however, such as one encounters often enough in acute uræmia, I have only observed in this single case.

While lying in a stupefied condition, these patients pass their fæces and urine under them. But even previously to their insensibility and state of uncleanness, the emanations derived from these chronic uræmic cases, whether from the breath they expire or from their perspiration, frequently have an excessively repulsive *urinous smell*. This symptom may perhaps depend upon the clammy sweat with which towards the end of life their skin is usually bedewed, and by which the epidermis, previously for the most part dry, comes to be fairly macerated. Under these circumstances it may happen that the skin of the face, of the neck and upper parts of the chest, and in males, too, especially the hair of the beard, becomes covered over with innumerable *crystals of urea.* The urea is excreted in the sweat of approaching death, and crystallizes upon the skin after evaporation of the water. In one case I saw the entire full beard of a patient, who lay in a state of coma, covered with tufts of such needle-like crystals; his beard looked as if it had been white frosted, and as I came into the ward I thought the barber must have left him after soaping his face to shave him. In another instance, two days before death, I saw the face and the skin of the trunk covered with crystals of urea.

Lastly, I must mention the occurrence of *hemorrhages* as a by no means rare event in the latter stages of contracting kidney, and one, too, by which the fatal issue may be induced. These bleedings occur most often from the nose, and I have known them in one case to happen repeatedly for fully a year before death. But besides this I have seen subcutaneous effusions of blood occurring principally in the extremities, partly in the form of petechiæ, and partly in the form of ecchymoses (vide Cases VIII. and XXI.). Hemorrhage may also arise from any other mucous surface, from that of the mouth, the stomach, the intestines, or the bronchi. Indeed the mucous membrane of the urinary and sexual channels are the sole ones from which I have never seen similar blood discharges take place towards the end of life. Some of my patients, at the date when the bleeding first happened, were in a good state of nutrition and of strength; but all of them died before the expiration of two weeks; the only exception was the case mentioned above of repeated nose-bleedings. He was a man seventy years of age, very thin, but for his age still vigorous, who except for his nose-bleeding had nothing whatever to complain of, and had never exhibited a trace of œdema. It was not until he evinced slight anasarca of the ankles, after having had these repeated attacks of epistaxis, that I examined his urine, and in this way discovered his malady.

Here again the most important evidence we obtain, and that which brings us to the clearest understanding of the whole train of the symptoms, is derived from *accurate examination of the urine*, and from careful estimation of the amount of functional incapacity exhibited by the kidneys during the process of their contraction. Only, one must not fancy that this information is to be obtained by the analysis of single samples of urine. There exists no kidney malady in which the danger of deceiving oneself is so great, if only a few, and these incomplete, examinations of the urine are instituted. *For it is just in these very cases of genuine contracting kidney that urine is occasionally and temporarily excreted, which is in no way to be distinguished from that secreted by healthy kidneys.*

In the well-marked cases, however, the deviation of the renal functions from their normal standard is so characteristic that the affection can be recognized by examination of the urine alone, and this although no other symptom may seem to point to this malady.

In the first place, *the quantity of urine secreted* during the twenty-four hours is *always remarkably increased*, so that these patients urinate far more abundantly than sound persons of similar form and build, unless these be excessive drinkers. It is self-evident that a patient who passes a great deal of urine is necessitated to repair this loss by the ingestion of a large quantity of fluid. Hence arises the very common mistake, above referred to, of these patients supposing that they have diabetes because of their thirst and excessively frequent micturition.

Case XXII.—The most extreme instance of polyuria I ever saw happened in the case of a gentleman, forty years of age, who had run away from the severe winters of his native place in Eastern Prussia, on account of an obstinate bronchial catarrh. I came across him at Wiesbaden, where he consulted me one day. Even before he set out on his journey he had noticed that his sight was bad, but while on the way he became so blind as hardly to be able to find his way about in the streets of a town which was strange to him. Up to this date he had lived in his ordinary way, and much according to his own inclinations, not shunning exposure of any kind, especially quenching his enormous thirst, for instance, with wine, and beer, and seltzer water, in just such quantities as were required. Neither was he now in the least inclined to put any measure of constraint upon his habits or desires, for, apart from his thirst and blindness, he felt perfectly strong and well. The pulse of remarkably high tension, and the evidence which existed of considerable hypertrophy of the left ventricle, directed me to the true cause of his malady. With the assistance of my colleague, who had called me to him, and who was his close neighbor in the hotel, I succeeded in measuring the entire quantity of urine which he passed during a night of twelve hours' duration, *i. e.*, from 8 P.M. to 8 A.M. This consisted of 6,000 c.c., it had a specific gravity of 1004 and contained albumen. The patient declined any further examination of his case.

Such an extreme increase of the urine as this in contracted kidney is certainly rare, and quite rare in patients in a hospital, who are subjected to certain rules and control in their entire diet scale. No other patient of mine ever passed such a huge amount of urine as my Wiesbaden friend. An exact estimate of the amounts passed in these cases during any considerable period of time, is, moreover, difficult to effect under any circumstances other than those presented in a hospital.

In only one private case could I ever feel sure that I had really estimated the entire quantity of urine passed *for a whole month.* This patient excreted on an average 3,350 c.c. daily. The smallest quantity passed on any day in this month was 2,520 c.c., the largest was 4,805 c.c. In another case, treated in the hospital, the daily mean average amount of an observation, extended over six months and involving seventy-six measurements, was 2,200 c.c. per diem. The maximum passed was 4,200 c.c., and the minimum 1,500 c.c. during twenty-four hours.

So excessively profuse a secretion as this of course compels the patient to urinate frequently, and here too the fact is remarkable that the patients are invariably more tormented with the desire to pass water *by night than by day.* One of my private patients, for example, who throughout his day's work from 9 A.M. to 4 P.M. had no call to empty his bladder, was forced to get up three or four times every night to urinate. It appears that this *greater frequency of the desire to micturate at night* is founded upon the *more abundant secretion that takes place at this time.*

The diurnal amount of urine passed by the above-mentioned patient was collected for a month, and upon twenty-six days out of this month the night urine was separated from that of the day. The day's urine, taken from seven in the morning to ten at night, stood at an average at 1,370 c.c. (91 c.c. per hour, that is), while the night's urine, consisting of that passed during the nine night hours, presented an average of 2,190 c.c. (or 242 c.c. per each hour). This patient was forced to get up at least four times each night to pass water. This circumstance, namely, the night urine exceeding the day urine in quantity, certainly very often occurs in this disease. Thus one of my hospital patients passed in ten successive nights, upon an average, 960 c.c. each night, and upon the corresponding days only 635 c.c.

For all this, such an abnormal distribution of the main energy of the functional activity of the kidney over the different periods of the twenty-four hours is by no means invariable in granular atrophy. Other cases of my own (in my hospital practice), tested in this particular direction, showed just the same conduct of the kidney in this respect as obtains in healthy individuals, namely, that more water was excreted by day than by night.

The great majority of patients whom I have been able to watch closely have completely confirmed by their proceedings the general rule that *the genuine process of contraction of the kidney is associated with polyuria. But neither does this symptom at once become prominent at the very beginning of the malady, nor does it persist absolutely to the very end of the case. In fact, in the progress of the disease it may entirely subside for a longer or shorter interval of time*, whenever, from any debilitating influence, the vigor of the heart's propulsive powers is diminished or affected for a longer or shorter period. Indeed, under such conditions we find that the urinary secretion of these patients falls back to an abnormally small amount.

The last patient above mentioned, whose day and night urines were separately estimated for ten days, may serve as an example of a case of granular kidney at the commencement of its development. As the result of twenty observations, he passed on an average 1,590 c.c. of urine in the twenty-four hours. Again, in a broken-down drunken subject, who was under my care during the last month of his life, the diurnal quantity was reduced to 1,000 c.c. H. H., the patient aged eighteen (vide Case XXI.), passed, the month before her death from uræmia, a daily average of 990 c.c. of urine upon eight successive days. In the last fourteen days of her existence diarrhœa set in and rendered the collection of the entire urine impossible. A girl of twenty, C. Kr., who had an attack of endocarditis in the course of her renal malady, passed, upon an average of twenty measurements effected during the last month of her life, only 763 c.c. daily.

In point of fact, *the urinary secretion may be completely arrested for several days before death* if the patient loses much water by any other channel, as by diarrhœa, or if symptoms of severe collapse precede death for some time. Indeed, the quantity of urine secreted per diem in genuine contracting kidney disease is subject to far larger fluctuations than occur in the inflammatory renal processes previously described. These variations in its amount are not merely due to intercurrent disorders of the circulation and to febrile complications, but occasionally depend entirely upon purely neurotic conditions.

The above-mentioned merchant, L., who had been passing a daily average of 1,500 c.c. of urine for the month during which he was under observation, received on the 30th of November some shocking intelligence, which provoked the most extreme anxiety in him. On the 2d of December he passed only 500 c.c. of urine. On the 5th of December, on the other hand, when his anxiety was overpast, the amount for the entire day reached 7,500 c.c.

The *color* of the urine, corresponding with its large amount, is as a rule *pale yellow*, or more often *yellowish green*. Generally it is *clear*, and in most cases it *deposits no sediment*. It is certainly a most rare event for it to deposit a sediment of urates upon cooling.[1] Much more often one finds at the bottom of the chamber utensil some separate *crystals of uric acid*.

Just as the ordinary color of the urine in contracting kidney is pale, so its specific gravity is, as a rule, low, and corresponds to the amount of fluid passed. *But in this form of renal disease, too, we perceive that the specific gravity of the urine depends upon the rapidity with which the secretion is being effected.* However, as a rule, we can state that the specific gravity of the urine remains below normal in this complaint. Thus, for months and even years, examining the water nearly daily, I have found the specific gravity of the urine of such patients excessively low, and fluctuating only within very narrow limits, varying, for example, between 1004 and 1012.

At the commencement of this renal malady, and so long as the polyuria is inconsiderable, the small concentration (or low specific gravity) of the urine may not attract attention.

The young blacksmith, A. L. (*vide infra*, Case XXIII.) passed the daily amount of 1,000 c.c., having a specific gravity of 1022, and some portions of it rising even to 1027.

---

[1] While the non-deposition of urates in the urine of these cases of granular kidney is the almost invariable rule, as Bartels here clearly states, a case which came under my care in November, 1876, illustrated the circumstance that urine may deposit urates in a most advanced case of granular kidney, both organs being reduced to as tiny dimensions as I have almost ever seen. The case was complicated by cirrhosis of the liver and pericarditis. The specific gravity of thirty ounces of urine passed on two days amounted to 1028, and the deposition of urates upon cooling was considerable enough to deceive me as to the state of the kidneys.—TRANSLATOR.

Even in far *advanced cases* of contracting kidney circumstances may arise to provoke the passage of urine of high concentration.

Thus H. H. (Case XXI.), when three weeks before her death she had just got over her first attack of uræmic convulsions, passed during the succeeding twenty-four hours only 525 c.c. of urine, having a specific gravity of 1023. Later on, again, when the diurnal quantity rose once more to 1,500 and 1,700 c.c., the specific gravity fell to 1012 and 1009.

Should the process of contraction, however, have advanced up to a certain extreme limit, it appears impossible for the urine, under any circumstances, to attain such a concentration and specific gravity as it ought normally to present.

Numberless observations made by me upon patients who remained under my care up to the end of their lives, convinced me that *in extreme cases of this nature, even although the amount of urine passed per diem fell to a few hundred cubic centimetres only, the specific gravity could not rise any longer over* 1009 *or* 1011. The lightest urine I ever examined in a genuine case of contracting kidney had a specific gravity of 1004, and was derived from my Wiesbaden friend previously spoken of.

Testing the urine chemically we find that it gives, as a rule, a feebly acid reaction. Should it be alkaline, this is either the result of decomposition that has occurred since its excretion, or the consequence of some dietetic or medical treatment, or perhaps is derived from the complication of some bladder affection, as already noticed, and which is by no means very uncommon. In a description of the chemical constitution of the urine derived from kidneys in process of contraction, the greatest importance will attach once more to *the quantity of albumen contained in the secretion. But albuminuria*, as later experiences in this direction have taught me, is *no constant symptom in this affection.* Tuengel[1] already announced in 1861 that he had been led into error by the absence of albumen in the urine of such patients, and had failed to recognize their disease ; but it has never been shown by either Tuengel or myself, or any other observer, so far as I know, that albuminuria can be absent throughout *the whole course* of the contracting process. *Certainly, albumen in the urine forms one of the characteristic symptoms of the disease.*

---

[1] Klinische Mittheilungen von der medicinischen Abtheilung des Allgemein. Krankenhauses in Hamburg. S. 77. 1861.

Sometimes, at the beginning of the disease, I have for a short time *failed to find* albumen in the urine, and in one case, in a subject who was very much reduced, it was absent for a greater length of time.

The temporary absence of albumen I have witnessed repeatedly and invariably under one and the same conditions. For purposes of diagnosis the matter is one which deserves to be detailed at greater length. I first had my attention directed to the alternating appearance and disappearance of albumen in the urine by the following case:

Case XXIII.—In March, 1872, a young blacksmith apprentice, A. L., twenty-one years old, came to my clinic on account of pains under his sternum and for frequent attacks of palpitation and oppression. Examining his chest, I found the præcordial dullness abnormally increased, especially so in the direction of the clavicle; the *systolic* sound at the *apex*-beat, and the *diastolic* sound at the origin of the *aorta*, were remarkably increased in strength, the latter being of a flapping nature. The pulse was tense and pressing, its frequency increased (from 90 to 100). The pale yellowish green urine contained albumen. The diagnosis of renal atrophy was made at the outset, and fourteen days later the patient was admitted into my ward. *Upon the following morning, when the urine which he had passed during the night was examined, no albumen was found in it. But it appeared again in the course of the day in the urine which he passed when he was out of bed and part of his time out in the open air, to disappear once more in the fluid he secreted during the ensuing night.* This alternate appearance and disappearance of the albumen in the urine continued throughout the entire long period of seven months during which the patient was in our clinic, according as he was out of bed and went about the ward and walked out-doors or kept himself quiet in bed. Nor did this variable result depend upon the period of day, for the urine remained free from albumen during the day-time, when, for experiment's sake, the patient was required to keep in bed by day. Towards the end of the time this patient was under observation, in October, 1872, the day's urine, too, contained scarcely a trace of albumen, so that I entertained the hope of being able to adduce a case of well-established (as I believe) contracting kidney cured, under my treatment; but my patient, feeling quite well himself, got out of patience and quitted the hospital.

Two and a half years have now elasped since I heard of him, and as he lived close to Kiel, I think this fact may be held to show that he has remained well up to this time.

Exactly the same constant alternation in the appearance and disappearance of albumen from the urine at the commencement of contracting kidney, according as the patient moved about or stayed in bed, I have observed since this in two other cases, which were also some time under treatment in our clinic, and which, in addition to albuminuria, presented the characteristic symptom, hypertrophy of the left side of the heart, in warranty of the nature of their malady. But, in order to be still more certain that we were correct in our interpretation of the facts observed, we made some examinations of the urine of another patient, who was under treatment in the hospital for Bright's disease and albuminuric retinitis, and who also had very considerable hypertrophy of his left ventricle.

In this instance both the night and the day urine invariably contained albumen, but with this difference, that upon the days when this patient walked about out of doors in fine weather (summer), the percentage as well as the absolute amount of albumen contained in the urine was considerably in excess of that furnished in the fluid secreted during the night. I will give a more detailed description of this in many respects interesting case.

Case XXIV.—Marx Misfeld, thirty-one years of age, a carter, had been attached to the army in the French campaign as gunner, and had never been ill. For some time he had noticed that he must get up at night frequently to make water. On the 23d of June, 1872, while he was occupied at his work, he was suddenly struck completely blind in his right eye. This lasted half an hour, and then his vision returned, but again half an hour later he became completely blinded in that eye, the sight of the left eye remaining unaltered.

On the advice of my colleague Voelckers, who had examined his eyes, M. was admitted into the medical clinical wards on the 26th of June.

State on admission.—Patient is small, but strong, well nourished and of fresh complexion, and feels perfectly well. The heart's impulse is heaving and forcible in the fifth left intercostal space in the mammary line. Above and to the left the præcordial dullness is somewhat increased beyond normal limits. The sounds are clear and loud, the diastolic sound over the aorta exquisitely ringing. Pulse of high tension, 90. The examination of lungs and abdomen elicited no abnormality. No œdema present anywhere. The result of the *ophthalmoscopic examination* made by Herr Voelckers is as follows: Slight neuritis of the optic nerve of both eyes; a few small white specks and little blood effusions in the left retina; in the right arteria centralis retinæ is an embolus. There is very little sensibility to light in the right eye; on the left side vision is unaltered.

The *urine* is pale, clear, with a specific gravity of 1007, is secreted in abundant quantities and *contains albumen*, but no tube-casts. These appeared, however, at later examinations as a constant ingredient, having the form of single, quite pale, hyaline casts.

The patient remained in hospital till July 31st. During this time his urine was carefully collected and measured. It proved, as *the average* of thirty-two measurements, that he had passed 1,900 c.c. of urine *daily* (the maximum being 2,600 and the minimum 1,215). The *specific gravity*, which was most frequently 1010, fluctuated between 1007 and 1013.

During this time observations were furthermore made with regard to the proportions of albumen found in the urine secreted during a state of rest and that secreted during bodily activity, and it appeared that both quantities during the first few days contained an abundance of albumen. On the 30th of June the daily quantity of urine was 1,770 c.c., containing four per cent. of albumen, or 7.08 grammes. When, on the 1st of July, the patient was confined to his bed, the amount of albumen at once diminished, and, indeed, fell to such a degree that in some single specimens the amount of albumen could not be quantitatively determined, as heat only caused a slight cloudiness of the fluid.

After he had been kept in bed for ten days, the proportion of albumen in the urine in twenty-four hours, which amounted to 1,585 c.c., had fallen to $\frac{84}{1000}$ of one per cent., or 1.331 grammes. When, after the 13th of July, the patient left his bed during the day, the per cent. of albumen in the entire quantity did not exceed $\frac{2}{10}$ of one per cent., hardly over two grammes a day. But the percentage of albumen was uniformly higher by day than by night. While the amount of urine passed during the day, as a rule, was greater than that during the night, *the proportions of albumen contained in the day and night urine respectively* fluctuated between a ratio of 1.5 to 1 and 3 to 1. The excess in the amount of albumen contained in the day urine was, however, still perceptible when, between the 22d and 29th of July, the patient was once more kept in bed both night and day, and during this period the amount of the albumen contained in the day's urine to that of the night stood in the proportion of 1.025 to 1 and 1.51 to 1.

On the 31st of July, as he was feeling completely well, M. was discharged, though the sight of the right eye was hardly at all improved.

On the 13th of September of the same year he returned to the hospital. Since his discharge he had been feeling so well as to have been able to follow his employment again. But early in September he suddenly fell insensible from the back of his horse. Sensibility did not return for several days. He had *right hemiplegia and incomplete aphasia.*

State on admission.—Evident emaciation since his discharge at the end of July; incomplete paralysis of the right leg; nearly complete of the right arm; paralysis of the facial nerve and of the hypoglossal upon the right side; slight anæsthesia upon the entire right side of the body. The aphasia which previously existed is decidedly less; still a few words are missing, as well as some letters in the alphabet.

His ideas are somewhat confused, still he voluntarily assures us that his speech has improved in the last few days.

Further examination demonstrated the fact that the area of *præcordial dullness* had become extended since July. The *apex-beat* could be seen as well as felt in the fifth and sixth intercostal spaces outside the mammillary line. The fifth rib was plainly lifted up by the systole. The *diastolic sound over the aorta* was much increased. Pulse tense, 80 to 90.

Lungs and abdomen showed nothing abnormal. *The urine* was passed in abundance, was clear and of low specific gravity, containing some albumen, and some few perfectly pale and narrow casts; no fever; vision as before.

Up to the beginning of January, 1873, the patient's condition steadily improved; he gained especially in the power of moving his paralyzed limbs, while his aphasia remained as noticeable as before.

On the 6th of January the patient stayed in bed, complaining of ringing noises in his ears and of black spots before his eyes; his anæmic aspect could not fail to attract attention. Upon closer inquiry it appeared that for the last three days he had *lost a good deal of blood* each morning at stool; and in the night-vessel, which had been used shortly before, we found from 400 to 500 grammes of bright red blood partly coagulated. The rectum was suspected to be the seat of the hemorrhage, and for its more thorough examination the patient was transferred to the surgical clinic. The examination here instituted gave negative results, and the patient was returned to the medical side again on the evening of January 10th, the hemorrhage having meantime stopped. The patient was now feverish. Temperature 39.2° (102.3° F.) in the axilla; pulse 116–120; ordered sulphate of quinine two grammes.

On January 11th the bleeding from the bowel began again, and was this time intractable; in addition he had troublesome pain in the abdomen; retention of urine necessitated the use of a catheter. The patient's strength failed; his temperature fell, and death ensued on January 14th.

Autopsy.—Skull-cap small, heavy, and thickened; dura mater pale and thicker than normal, but easily detachable even from the basis of the cranium; pia mater also easily detached.

Brain substance shining, everywhere firm and anæmic; ventricles small, their walls firm. *To the outside of the corpus striatum upon the left side was a cavity the size of a nutmeg*, filled with a somewhat brownish, turbid, but completely fluid substance. The walls of this spot were dark brown, and, especially towards their lower portion, were of a soft consistency. The cavity itself was surrounded with white brain substance, but it extended forward into the anterior lobe, and backward into the inferior cornu, in the shape of a fissure, the full length of which was 7 cms., its greatest width 2½ cms. The brain substance round about this fissure was colored brown, and softer than normal. Almost exactly corresponding in situation to this lesion on the left side, but situated a little deeper down below the corpus striatum on the right side, was a long cleft two centimetres long, filled with fluid exactly like that which was found upon the left side.

Upon removing the sternum the pericardium was largely exposed to view, but it contained only a little clear fluid. *The heart*, especially its *left ventricle*, was *enlarged to colossal dimensions.* It was 12 cms. in length, and 12 cms. in breadth; its walls 3 cms. thick; its muscular substance was exceedingly tough. The valves on the left side were strongly developed; their substance was firmer than normal. There was slight roughening upon the ventricular aspect of the aortic valves, and numerous yellow patches appeared on the inner surface of the ascending aorta. Right side of the heart somewhat dilated; valves tender; nothing wrong about the lung; in the abdominal cavity there was a small quantity of dirty, blood-colored fluid; spleen and liver normal.

The *left kidney* was 9 cms. in length, 3.5 cms. in breadth; weight 69 grammes. Capsule only firmly adherent in the upper two-thirds, where on its removal some portions of the renal substance were detached. The surface of the kidney in its upper part was very uneven, owing to the presence of fine granulations, but it was perfectly smooth and even in its lower portion. This lower smooth portion of the surface of the kidney stood out in prominent contrast to its upper half, being separated from this contracted part by a ledge-like boundary. The upper portion was very decidedly atrophied in its entire substance, but principally in its cortical part, so that the pyramids nearly reached to the periphery of the organ. Between the pyramids there was only a very small amount of grayish red cortical substance. As a whole the aspect of the kidney was rather anæmic than otherwise, its substance firm and tough. Pelvis of kidney narrow.

The *right kidney* was 8 cms. long, 4 cms. wide, somewhat thicker than the left, and weighed 73 grammes. Capsule everywhere adherent. The whole surface was uniformly covered with fine granulations. The cortical substance, rather paler than that of the left kidney, surrounded the bases of the pyramids generally with a broader border than was found in the left kidney. Its general consistency and other features resembled those of the other organ. The *stomach* was normal.

Intestines thin—their mucous membrane quite strikingly pale, only here and there a few small venous radicles filled with blood; no trace of ulceration anywhere.

The following case is the only one that has come under my observation in which albumen was entirely absent from the urine throughout, and where, therefore, the renal malady was not recognized during the patient's lifetime.

Case XXV.—A gardener, fifty-six years of age, from Kiel, who had led the life of an adventurer for some years, the greater part of his time having been passed in America, was brought into our hospital here at noon on January 26, 1873, in a state of complete unconsciousness. By chance I happened to be present at the time of his admission and examined him immediately myself. All that could be learned of the history of the case was that the patient had for some considerable time been a regular drunkard, and as a rule spent about half a thaler daily on common

Holland schnaps, but ate very little. It had struck the people at the tavern where he stayed that his strength had been declining visibly for the last few weeks. He would not go out at all, but came down of a morning from his sleeping apartment to the dram shop located in the same house, and had to be carried up again every evening, drunk. On the 25th of January he never made his appearance, and was found lying insensible on his bed, which was in a cold room.

Upon his admission on the 26th, we were struck by the *very low temperature* of his limbs, and by means of a thermometer introduced into the rectum found that the *temperature of the bowels* was 28.2° C. (or 83° F.). The pulse at the wrist was not to be felt, nor could the heart be felt.

But with the stethoscope one heard very faint *heart-sounds*, and we were able to count *fifty two contractions of the heart per minute.* Respirations sixteen. By dint of protracted hot baths at 40° C. (104° F.), we succeeded in gradually warming him so far that by the evening of the 27th of January the temperature in his rectum had risen to 37.8° C. (100.1° F.); but this always dropped down again directly the patient was put back into bed, after being for hours kept in the bath. It was not until the 1st of February that these warming baths could be discontinued.

The attempt was made on the very first day to draw off the contents of his bladder by a catheter, in order to get some of his water. But this was thwarted by a very high degree of stricture of the urethra, and on the following days all the urine was passed in his bed. It was not until the 29th of January that we succeeded in getting 425 c.c. of urine. *This was clear, rather dark brown, of moderately acid reaction. Specific gravity* 1013; *no albumen.*

As the patient recovered himself a little, it was apparent that he labored under delusions, and was constantly delirious. His temperature on February 2d went as low as 35 C. (96° F.), and even on the 7th stood at 36.4 (97.5) in the rectum. The pulse was 56 to 58. He began to have a keener appetite for the first time on February 7th, and from this date the temperature and pulse recovered their normal height, but the delirium still got worse, so that by night he often wandered about in the ward.[1] Under these circumstances it was excessively difficult to collect all his water, for when he went about some urine constantly dribbled away from his bladder. For all this, we succeeded in collecting nearly all that passed for four different days. The amount varied between 850 and 1,050 c.c.; the specific gravity between 1012 and 1018. *No albumen was contained in any portion of this, and there was so little urea that during no one day did this measure more than ten grammes.* On the 20th of February the patient was vaccinated to protect him from small-pox, at that time prevalent. Six perfectly healthy vaccine pustules were developed; but on the evening of the 26th of February fever set in with a temperature of 39.2° C. (102.2° F.). On the following day the temperature was maintained at over 40° C. (104° F.), and on March 1st even reached to 41.4° C., or 106.2°

---

[1] So far as I know, this is the only well-observed case in which an individual, whose temperature taken in the rectum had fallen to 28.2 C. (83° F.), has completely recovered and maintained his natural heat again for any length of time.

F. With the commencement of the fever his strength visibly declined; he passed both fæces and urine pretty regularly under him, and we could but rarely obtain even small quantities of his urine; *but this, since his fever began, contained small amounts of albumen.*

Under symptoms of increasing debility death ensued on the 3d of March, early in the morning.

The following facts only need be given from the record of the post-mortem:

Both kidneys were embedded in a very thick panniculus adiposus. The right one was *diminished in size*, measuring 10.5 centimetres in length. Capsule firmly adherent. Surface irregularly granular, covered with numberless large and small cysts. On section the substance of the organ was very pale, of a grayish red. Pyramids small, cortical portion relaxed. *Left kidney very small*, 8.3 cms. in length, 3.3 cms. in breadth, 2.3 cms. thick. Capsule everywhere firmly adherent, surface uneven, nodular, and presenting both large and small cysts. Bladder much distended with dark brown urine, acid, containing an abundance of sediment; but on heating this by boiling, or after the addition of acetic acid, it hardly became even cloudy. The walls of the urinary bladder were very thick, its inner surface covered with very prominent trabeculæ. Orifice of the urethra exceedingly small, frænum considerably distorted by cicatrices. Traces of old scarring on inner surface of urethra. No trace of œdema of the subcutaneous cellular tissue. There were a few drops of clear serum in the pericardium, pleura, and peritoneum. Heart very large, but very much relaxed. No cause of the febrile process he had developed was discovered in the body.

Under all circumstances, however, the amount of albumen contained in the urine in the course of genuine contracting kidney is insignificant in comparison with the high percentage furnished in inflammatory processes affecting the same organ. Indeed, one may say that the most albuminous urine furnished by the contracting kidney hardly, as a rule, contains so much albumen per 1,000 parts as that furnished by the inflammatory kidney per 100 parts. It is self-evident that the albumen varies in its amount considerably in the different cases and in different samples of urine from the same case, fluctuating between a scarcely perceptible cloudiness on boiling and an amount of 5 parts by weight of dry albumen to 1,000 parts by weight of urine. Exceptionally, for instance[1] after an attack of uræmic convulsions, I have nevertheless found enormous quantities of albumen temporarily to exist in the urine furnished by contracting kidneys, the amount reaching, for example, 2.3 per cent. (compare Case XXI.). But as a rule one finds only a few parts per 1,000. *The daily loss of albumen* sustained by patients of this kind *reduces itself*, in spite of the ordinarily profuse urinary secretion, in most cases to *an extremely small sum* per diem, a few grammes only or the fraction merely of a gramme. As a result of a large series of analyses which were instituted in our clinic here upon the urine of such patients, I will here introduce a few cases in brief calculated to throw light upon this subject, remarking at the same time that I have only selected those cases in which the diagnosis has been verified by post-mortem examination.

---

[1] The connection between epileptic attacks and the presence of albumen in the urine has been already noticed by me.

K., a brewer, forty years of age, passed, on an average of twenty-one analyses, conducted during a period of three months, 1.3 grm. of albumen daily. Three years later he died with uræmic symptoms.

B., a teacher of gymnastics, fifty-six years of age, upon a mean of eleven analyses, instituted at wide intervals of time during the last years of his life, passed 1.486 grm. He died of cerebral apoplexy.

The brewer M., aged thirty-four years, who died of hemorrhage from his bowels, passed, the half-year before his death, according to the mean of fourteen analyses, 2.163 grms. of albumen daily in his urine.

S., a laborer, thirty-two years of age, in the last six weeks before his death, which followed hemorrhage from the nose and bowels, with uræmic symptoms, passed, as the mean of thirteen analyses, 5.962 grms. of albumen daily.

Two young girls, aged respectively eighteen and twenty, died with uræmic symptoms shortly after their reception into the hospital, and after death their kidneys were found contracted in the most extreme degree. The total amount of urine was collected in the one case for three and in the other for four days, and analyzed. The first had been passing 2.4 grms., the second 1.85 grm. of albumen per diem.

The cases here placed together and those previously detailed will suffice to give a correct impression of the diurnal loss of albumen which patients experience in the progress of the process of contraction of the kidneys, through the abnormal conditions under which the urinary secretion then takes place. They show, too, that at the commencement of the malady the excretion of albumen is very insignificant—so much so that it is not every portion of urine that contains albumen at all; and that, as the disease progresses, the total loss of albumen sustained increases, until towards the end, as the powers fail, and the heart's contractile faculties too, the loss of albumen diminishes again, or it even may cease to be excreted altogether. They show, further, that only peculiar conditions, such as affect the general circulation, and which can be provoked by the severer kinds of epileptic convulsion, will lead temporarily to the excretion of as large amounts of albumen (percentage as well as total) as are ordinarily furnished in the parenchymatous inflammation of the kidney.

My own observations have further taught me that, *in one and the same individual, the amount of albumen contained in the urine in contracting kidney will be subject to great variations*, according to the mode of life, the food, and the general state of nutrition. People who are reduced and debilitated by privation and neglect apply to a hospital when they begin to become dropsical. They pass a very pale and exceedingly light urine, which has but little albumen in it. If, under good regime and careful nursing, these individuals pick up, the dropsy very rapidly subsides, and the strength may be recovered nearly as rapidly, so long as the renal affection is not too far advanced. As soon, however, as the patient begins to test his powers and to go about actively, etc., the absolute as well as relative amount of albumen contained in the urine increases again, as has been amply shown above. This increase in the total amount of albumen lost appears natural, because the total quantity of urine excreted, under the conditions here mentioned, is increased.

No constant ratio exists between the percentage of albumen and the total quantity of urine; still, as a rule, I have recognized a higher percentage of albumen as coinciding with notable decrease in the quantity of the urine passed during twenty-four hours. The following examples, however, illustrate how much this circumstance may be dependent upon the state of the organs of circulation.

The laborer St., a man of athletic build, was taken into the hospital on the 3d of June, 1868, on account of anasarca of his lower extremities. His complexion was pale, and he was much debilitated; *his urine contained albumen, and his heart was hypertrophied.* With rest and good care he picked up very quickly, so that in a very short time the œdema of his legs disappeared. During the six months he stayed in the hospital he rejoiced in an excellent appetite, and soon after his admission felt well every way. During the six months he was under our observation seventy-six complete analyses of his urine were made, which gave 2,200 c.c. of urine as the daily average excreted, containing 7.57 grms. of albumen and 33.38 grms. of urea. The *largest amount* of urine passed by him *upon any one day* during these six months was 4,200 c c., sp. gr. 1006, albumen 0.098 per cent., or 4.116 grms.; the *smallest* was 1,600 c.c., sp. gr. 1015, albumen 0.482 per cent., or 7.712 grms. Thus, in this case it appears that *the largest quantity of urine passed per diem contained, both relatively and absolutely, the smallest amount of albumen that was furnished on any day during the period he was under observation.*

On the other hand, the girl Kr. whose case was given above, and who suffered from an attack of endocarditis in the course of her renal malady, leaving her with mitral insufficiency, during the last month of her life passed only 763 c.c. of urine daily, as the mean of twenty analyses, and lost on an average 1.23 grms. of albumen per diem. Here *the largest quantity of urine excreted on any day* was 1,180 c.c.; sp. gr. 1011.5.; albumen 0.18 per cent., or 2.12 grms.; and the smallest quantity was 420 c.c.; sp. gr. 1011; albumen 0.12 per cent., or 0.504 grms.

In both these cases the state of the circulatory organs unmistakably made itself felt in influencing the amount and characters of the renal secretion. In the robust man the increased blood-pressure in the arterial system, resulting from the hypertrophy of the left ventricle, produced polyuria and an abundant excretion of albumen from the affected kidneys. In the girl the insufficiency of the mitral valve rendered the previously hypertrophied heart powerless to maintain the requisite tension in her arteries, and thus the urine previously so abundant fell below what was normal, and the quantity of albumen contained in it became insignificant.

* * *

Corresponding to the low specific gravity of the urine in contracting kidney, *its percentage of solid constituents, and especially of urea, is abnormally small;* and it stands to reason, therefore, that the percentage of solids, like the specific gravity, will vary inversely with the rise and fall of the diurnal amount of urine.

The laborer St., previously noticed, passed as the largest amount of urine among seventy-six separate analyses 4,200 c.c., having a sp. gr. of 1006, and containing 0.6 per cent. of urea. The highest urea excretion amounted to 2.1 per cent., and occurred when the quantity of urine passed per diem was respectively 2,075 and 2,100 c.c. (and when the sp. gr. was 1013 and 1019).

When I reckon up *all the cases* in which I possess analyses of the urine, and in which the diagnosis was confirmed by autopsy, I find that *it is an exception to the rule to meet with more than* 2 *per cent. of urea in the urine of contracting kidneys*, and that an amount exceeding this occurs but rarely even in patients whose renal malady has been recognized for years before their death. As a rule, in well-marked cases, *the percentage of urea stands between* 1 *and* 2 *per cent.*, and occasionally even falls *below* 1 *per cent.* But in the larger number of instances the sensible deficiency in the urea dependent upon the small percentage amount in which it is excreted is concealed for years by the polyuria associated with the malady.

The brewer K., æt. forty, three years before his death excreted 19.24 grms. of urea as his daily average; the teacher of gymnastics B., in the last year of his life, 22.53 grms. The brewer M., aged thirty-four, passed 26.94 grms. six months before his death; the day-laborer S., 26.47 grms. The case of the laborer St., already mentioned, who passed a daily average amount of 33.83 grms. of urea, reckoned from seventy-six analyses and extended over a period of six months, shows that if the general state of nutrition be good, the appetite excellent, and the food healthy as well as abundant, the excretion of urea through contracted kidneys may reach and be maintained at quite a high measure.

In individuals who are much reduced in their general nutrition the total amount of urea excreted daily is materially diminished, and that for obvious reasons; and the same thing holds true when intercurrent disorders of the general circulation effect a diminution in the arterial tension.

As examples of cases of this first kind, I have adduced three of extreme atrophy of the kidneys affecting three young girls, whose respective ages were twenty-two, twenty, and eighteen years, and who excreted in the last weeks of their lives daily averages of 16.3, 12.5, and 8.8 grms. of urea.

Kr., the girl of whom I have so repeatedly spoken, whose renal affection was complicated by mitral insufficiency, passed *during the last month of her life* an average of only 12.68 grms. of *urea daily.*

I have paid less attention to the other constituents of the urine in these cases of contracting kidney than to the abnormal and anomalous substances hitherto referred to. Still, in every case in which the urinary secretion has been analyzed quantitatively for the amount of chlorides and phosphates which it contained, I have found these substances to be present in less than the normal proportions. The total quantity of chlorides excreted in the urine may indeed on particular days considerably exceed the sum which sound kidneys excrete per diem in individuals upon an ordinary diet. But in those who are already much weakened, the daily amount of these substances excreted is far below the normal average.

The girl Kr., for instance, excreted as the mean of eighteen analyses instituted during the last months of her life only 1.7 grm. of chlorides, and 0.544 grm. of phosphoric acid.

The notorious dependence of the excretion of these substances by the kidney upon their ingestion in food renders the result of further investigations in this direction of no great value towards determining the question at issue.

In the great majority of instances of contracting kidney we find *uric acid* in but small quantities in the urine; in very dilute specimens we can occasionally obtain a mere trace of uric acid, precipitated by the addition of hydrochloric acid. This, however, certainly does not depend upon the state of the kidney; for in the urine of a case of old chronic arthritis which was itself albuminous and furnished by kidneys which were implicated in regular contracting disease, as proved by an autopsy made a few months later, I one day found a thick lateritious sediment with large grains of urinary gravel in it, and thoroughly well formed crystals of uric acid.

The urine furnished in regular contracting kidney disease, as a rule, deposits only very small amounts of sediment, and if secreted in abundance, as it ordinarily is, absolutely none at all.

For the microscopic examination of this sediment, in order to make it of any avail, the supernatant urine must be very carefully poured off from the vessel, and the sediment at the bottom should be collected in a proper funnel-shaped or tall champagne glass. Here, after a while, a faint gray cloud will sink to the bottom, and after pouring off the fluid the last drops at the bottom may be used for examination under the microscope. I prefer this mode of collecting the formed elements from the urine to a filter, since by this last plan the microscopical preparation is often so dirtied with fragments of fibre from the paper.

In a sediment prepared in this way one finds a few *scattered casts* under the microscope, side by side, perhaps, with some crystals of uric acid or oxalate of lime. But these casts are so rare that we often have to put up a good many specimens and search a long while before discovering even a few of them. Most of them are of the *narrow variety* and *perfectly hyaline*, or else lightly stippled with a few dark granules or finest fat-globules. It *rarely* happens that a *broad cast* is encountered, and still *less usual* is it to find a *dark granular cast. I cannot remember ever having found waxy, refracting, yellow-colored casts in this kind of urine.*

I found narrow *hyaline casts in large numbers* in the urine of the teacher of gymtics B., *after an attack of uræmic convulsions*[1] which he went through with five months before his death. In later examinations I had great difficulty in finding any, and then only a few. I found a *larger number of broad and dark granular casts in the single instance of the laborer S.*, five weeks before he died of hemorrhages from his nose and intestines. These forms, however, disappeared a few days after his admission into the hospital; and afterwards we only found narrow and hyaline casts in moderate quantities.

---

[1] This observation recalls one like it made by Huppert in an epileptic case. After severe epileptic fits, he found not only albumen invariably in the urine, but hyaline casts, too, in the specimens passed within the first three or four hours after the fit.—*Virchow's* Archiv. Bd. 59.

Isolated epithelial cells from the renal tubes are seldom presented in the sediment of this urine; but one discovers somewhat oftener a few healthy-looking epithelial cells, with distinct nuclei clinging to the casts, both to the narrow and to the broad ones; but there are never many of these, and seldom any cells or fragments of cells in the course of fatty degeneration.

Occasionally, too, small as well as larger *octahedral crystals* of *oxalate of lime* are seen attached to the tube casts. Sometimes we find a *few scattered red blood-corpuscles* in the sediment of this urine. But any *quantity of blood* mixed with the urine, or enough to bestow the recognized sooty red brown (toast and water), crumb-like sediment at the bottom of the vessel, is a thing which I have only seen in the case which was complicated with endocarditis, and to which I have frequently adverted. White *blood-corpuscles* or pus-cells, which in some cases are present in some abundance, will always have been added to the urine from some one of the mucous tracts belonging to the urinary passages; at least, one invariably finds epithelium derived from the pelvis of the kidney, or from the ureters or bladder, side by side with them.

Except in one instance, I have only instituted an *examination of the blood* of patients suffering with contracted kidney quite in the last stages of this malady. In every case I procured the blood by means of small venesections, made during the life of the patient.

The chief feature to which I directed my attention was the *specific gravity of the blood-serum.* In only one case the specific gravity of the whole mass of the blood was ascertained. This was taken in the case of the laborer S., from blood drawn by venesection five hours before his death; the specific gravity was 1050.

The specific gravity of the serum in the different cases is expressed in the following table:

Description of Individual.	Sp. Gr. of Serum.	Amount of Water in Serum.	Remarks.
Laborer, S.	1030.58	86.69 %	Venesection 5 hours before death by hemorrhage and uræmia.
Girl, H. H., dropsical.	1022.80	91.16 %	Venesection the day before death by uræmia.
Girl, Kr., dropsical.	1023.40	87.15 %	Venesection shortly before death by uræmia.
Girl, C. L., dropsical.	1021.00	88.77 %	Venesection the day before death by uræmia.
Laborer, H. H., drunkard, dropsical.	1024.80	90.66 %	Venesection one year before death, undertaken for the relief of uræmic convulsions.

I have several times been able to show urea obtained from the alcoholic extract of the blood of such patients as these, in the form of nitrate of urea, and once in notable quantity; but in the larger proportion of cases thus tested for urea the blood gave negative results.

My own observations upon the blood certainly admit of my arriving at no conclusions upon the nature of this fluid in the early stages of the complaint, for such examinations as were made were instituted upon the blood of persons in whom the renal atrophy was excessive, or who were reduced in their general nutrition, like the drunkard last referred to, by some other means. It is worth noticing merely that the last four cases concur in exhibiting low specific gravities in their blood-serums as well as dropsy.

But from the excellent state of nutrition and undiminished strength which so many individuals enjoy for years after their renal disease has begun, we must conclude that the blood preserves its normal properties for an equal length of time, and that its normal constituents are combined in their normal proportions in spite of the kidney affection. It is not until the kidneys are reduced to a state in which they can no longer fulfil the functions allotted to them, and when, therefore, retention of both water and urinary elements takes place in the system at large, that the blood appears to be more aqueous than is normal and to be contaminated with the constituents of the urine.

Review of the Post-mortem Appearances.

Genuine contracting disease affects both kidneys and both organs equally in most cases, although instances do occur in which one kidney is altered to a much greater extent than the other, and other instances in which a portion of one kidney may suffer a graver degree of change than the rest of it. The result of these changes in well-marked cases is a very considerable reduction in the volume of the two glands, so that in extreme cases they do not appear larger than children's kidneys. It is the cortical substance which is the principal seat of the atrophy; an atrophy so considerable that in some cases a quite small margin of cortical substance, hardly measuring more than a few millimetres in depth, surrounds the bases of the pyramids. The medullary substance itself, however, does not escape atrophy; its cones shrink into very narrow volume, and on section appear to be closely packed together through the wasting of the intervening glandular substance.

The pelvis of the kidney is occasionally distended to a pouch of pretty capacious size, but it is oftener drawn together into narrower dimensions than normal, and this appears to be the rule. As a rule, there is no change in the larger vessels entering the hilus of the kidney. The capsule is tougher and thicker than normal, and adheres firmly to the surface of the organ so that it can be separated from this only with difficulty. Portions of the renal substance are always found adhering to the capsule.

The surface of the gland, laid bare by removal of its capsule, presents an uneven appearance, owing to the presence of a number of tiny nodules (granules) which appear to be scarcely as large as millet seeds and are pretty uniformly of the same size. Among these, in many instances, we find cysts filled with clear contents, sometimes in large numbers, many of them no larger than the solid granules, but some as large as a hemp seed and even larger.

These small kidneys are remarkably tough, and offer considerable resistance to the knife upon section. The cut surface, too, occasionally presents a number of small cysts situated in the scanty cortical substance.

The color of the kidneys is not alike in different cases. Sometimes one finds these glands dark colored throughout, reddish brown; in other instances they may be strikingly pale, looking nearly grayish white; this difference is evidently due to the different amounts of blood contained in the kidneys of the dead body in different cases. In no instance, however, do kidneys of this kind exhibit that marked yellow color in any portion of their substance which distinguishes the remnants of kidneys in a state of fatty degeneration in secondary contraction after nephritis.

The microscopic examination of kidneys thus altered by granular atrophy shows that the diminution of the organ is attained by extensive wasting of the glandular structures proper, the renal tubes with their epithelium and the vessels attached to them. Between extensive layers of very firm and chiefly fibrous organized connective tissue are found some well-preserved renal tubules, lined with normal epithelium, and having healthy Malpighian tufts attached to them; but such tubuli are only few in number and mostly widely separated from each other, or else are distributed in patches by themselves. Whether the lines of striped tissue found in the interspaces are correctly described as consisting of the remnants of the membranæ propriæ of urinary tubules, deprived of their epithelium and therefore collapsed, is a question which I leave undecided. One very remarkable microscopical appearance is the great number of wasted glomeruli which appear like dark round bodies, and in which the outline of the capillary coils is still plainly perceptible; they are often grouped closely together, and appear much smaller than the normal Malpighian tufts which still remain; they lie in the midst of the striped and fibrillated connective tissue, perfectly independent of the tubuli uriniferi. Round about these remnants of wasted capillary tufts one recognizes fibrous tissue bands, which run concentrically round the glomeruli and unite in forming a thick, close-fitting capsule around them.

Wherever the little cysts above described present themselves in the cortical substance, their walls appear to be a homogeneous membrane, which is often strengthened by external fibrous layers ; their contents consist of a perfectly structureless, transparent, gelatinous mass. These cysts appear to be developed from partially dilated and strangulated renal tubules, the contents of the tubes having subsequently suffered *colloid* metamorphosis.

Grainger Stewart[1] is of the opinion that some of these cysts proceed from dilatations of Bowman's capsules, for in them he found the compressed capillary tufts lying pressed up against one side of the wall of the cyst. Klebs[2] has observed the same thing. This author, too, in support of the idea of the formation of the other cysts from strangulated portions of the curling tubuli, adduces their orderly arrangement next each other, like strings of pearls. In the colloid material filling the cysts Klebs was often able to recognize the boundaries of the particular cells from which these masses had proceeded.

The granules upon the surface of the kidney appear, under the microscope, as remains of renal tubuli still beset with epithelium. Here and there one sees this epithelium in a state of fatty degeneration, but it is more usually perfectly normal. Nothing abnormal is traceable in the straight tubes in the pyramids.

Such a state of general degeneration as we have here described is by no means arrived at in every case. I have examined kidneys where only partial contraction had taken place. This may occur as follows : one part of the kidney (its upper or lower end, for example) may retain its perfectly normal appearance, while the whole of the rest of it may be contracted. In such cases I found that the capsule was easily detached from the sound part, but adhered firmly to the diseased half. The sound part presented a perfectly smooth surface, the diseased portion being granular and knobby. The healthy portion was prominently raised on the surface of the organ, and stood above the level of the diseased parts (compare Case XXIV.). In other instances the contraction has been found to occur in patches, in which case it invariably commenced at the hilus of the organ, so that the surface of the kidney was to a greater or less extent depressed, and this irregularly, smooth insular portions being found standing out upon the surface, and being surrounded by other nodular depressed parts which lay between them. In these forms of the contracting disease the process was always farthest advanced round about the hilus, the upper and lower extremity of the organ as well as its convex border having suffered much less, and in these parts were found the largest islands of well preserved renal structure. But in all these cases the total volume of the kidney, although thus only partially contracted, had suffered considerable diminution, and the individuals, too, in whose bodies I found these changes, had experienced during life the clearest symptoms of renal contraction. The last case of this kind which I saw was in consultation with the late Dr. Th. Simon, in Hamburg, to whose kindness I am indebted for sending me afterwards this description of the post-mortem appearances:

---

[1] L. C. p. 182.

[2] L. C. S. 664.

> Right kidney the size of a nut. Left kidney evidently had formerly been hypertrophied, and was over five inches long. Its lower portion was correspondingly thick; but the upper part, and especially the middle portion, from the pelvis of the organ outward, was the seat of advanced contraction. Hypertrophy of the left ventricle.

It is just in such cases as these of *partial contracting kidney* that both kidneys are not always equally diseased. I have thus seen one kidney completely contracted and found the other perfectly sound in its lower quarter; and, again, one kidney contracted in its lower half, while the upper half and the whole other organ were in a condition of inflammatory swelling.

Aside from these changes in the kidneys, when both organs are affected and highly diseased, there is *but one other almost constant pathological condition* found in the body after death, namely, *hypertrophy of the left ventricle* of the heart. Every other organ may be perfectly normal, and in far the larger proportion of the cases there *is not a trace of dropsy present.*

A very ordinary feature of this disease, and one which may certainly be designated as a sequel to the renal affection, is the peculiar *alteration of the retina;* it occurs very often in both eyes, and is noticed more invariably than any other complication. This is the cause of the disturbance of vision which affects so many patients in the course of their renal malady. I append below (p. 476) a special description of this from the pen of my colleague Voelckers. A further very ordinary event found after death is a *considerable thickening of the skull cap.* The entire skull case is more massive and heavier than is normal. The thickened dura mater adheres firmly to the inner surface of the cranium, and the furrows which exist for the arterial branches on its internal aspect are deeply grooved, the diploë substance of the bone itself being remarkably thickened. Here, no doubt, is evidence of a hyperostosis affecting these bones.

It is not unusual to find *apoplectic effusions* in the substance of the brain, often quite extensive, while the blood effused may or may not have broken through into the ventricles. I have seen enormous destruction of brain tissue, through extravasations, in the bodies of persons who have died of this renal disease.

Lastly, I must mention the *dropsical effusions* which one finds in some bodies, in the cavities as well as in the meshes of the intercellular tissue, and which are attributable to the renal affection. This dropsy is seldom of any extent, and only quite exceptionally reaches to the amounts which we meet so commonly after parenchymatous nephritis.

The *general condition of nutrition* throughout the body may be perfectly normal, so that we cannot regard loss of fat or wasting of muscle as necessary accompaniments of this renal disease; although it is perfectly true that the self-same malady, in some cases, when it reaches, so to speak, its extremest grade, and when the functions of the gland substance are completely exhausted, does lead to general marasmus. But in a very decided majority of instances the affection proves fatal, either by uræmia or cerebral apoplexy, or by some final acute inflammatory effusion into the pleura, the pericardium, the abdominal cavity, the lung tissue itself, the skin, or the intercellular tissue, long before the final limit of destruction of the kidney is reached.

Inflammatory exudations are among the more frequent appearances found in the bodies of these patients, and may, under these circumstances, be looked upon as having been the immediate cause of death. The grounds upon which I consider these inflammatory processes, occurring as they do in the most different parts of the body, as the direct consequence of the renal malady, have been already given above. The endocarditis which affects the valves of the hypertrophied left ventricle may perhaps require some different explanation. This I have observed twice in the course of contracting kidney.

All other pathological changes which present themselves side by side with genuine contracting renal disease must be regarded as accidental complications of the complaint; at all events, I can adduce no others which deserve to be placed in a causal relation to it.

Upon the *exact nature* of the renal degeneration above described and its mode of origin, opinions are divided. Most of the German writers regard the state of kidney here described as the issue of parenchymatous nephritis, and identify it with that secondary contraction of these organs which ensues after nephritis. Since the writings of A. Beer and Traube, the term "interstitial nephritis" has come to replace that of "parenchymatous nephritis," the adherents of this doctrine believing that the changes which take place in the renal epithelium are under all circumstances of a secondary nature, and assuming that a general swelling of the entire intertubular connective tissue due to hyperplastic growth of the same precedes the diminution in size of the organ. This opinion has been accepted by the more recent French writers, Lecorché and Kelsch. This view was first opposed in England. Johnson[1] declared as early as 1859 that the small contracted kidney had never passed through an anterior stage of swelling. According to him the contraction is brought about by the primary destruction of the renal epithelium that lined the tubuli uriniferi. These cells are loosened from their attachment to the walls of the tubules, and are swept away with the urine; once completely emptied in this way, the tubules collapse. According to Johnson we have not, as the German and recent French writers, and Dickinson among the English writers assumed, to deal *with an absolute increase of the intertubular connective tissue, in the contracted kidney (provoked by anterior processes of inflammation), but with a merely relative preponderance of the same, as the consequence of desquamation of the epithelium of the renal tubules.*

---

[1] **Med. Soc. of London. Lancet, July 9, 1859.**

Grainger Stewart occupies an intermediate position in the view he takes of the matter, for he concedes the increase of the connective tissue, but will have it that this arises simply from its hypertrophic growth. He denies to the process the character of inflammation, since his microscopical examination of kidneys in advanced stages of contraction has failed in ever discovering to him a trace of free exudation into the stroma of the organ. He considers the destruction of the epithelium to be secondary, and the result of the growth of the interstitial tissue; he was struck, too, with the thickening of the walls of the smaller arteries in the diseased parts of the kidney.

Liebermeister, in his work "On the Pathological Anatomy and Clinical History of Liver Diseases," published at Tübingen in 1864, advanced exactly the same opinions even before Grainger Stewart, and had already proposed the same name which Grainger Stewart applied to genuine renal contracting disease, namely, "*cirrhosis of the kidney*" (p. 75).

Gull and Sutton, in their work cited above, refer the process of genuine contraction of the kidney to the changes produced in the arteries and capillaries. According to them, the thickening of the tunica adventitia of the smaller arteries narrows the calibre of the vessels and eventually leads to their complete obliteration. This thickening is brought about by a new growth of fibrous connective tissue. This connective tissue grows round about the capillaries too, although here still retaining its hyaline features, or else assuming the aspect of granulation tissue. The process does not affect all the arteries of the diseased kidney, nor every branch of the same arterial stem, to the same extent. It originates in the most superficial layer of the renal substance — that which borders nearest to the capsule — and spreads gradually more deeply into the cortical substance between the curling tubules. Proceeding from the sheath of the vessels the fibrous connective tissue grows round about the tubuli uriniferi, which are often only recognizable in the centre of concentrically arranged fibrous tissue cells by the atrophied remains of epithelial cells in their narrowed and compressed interiors. The renal tubes, together with their epithelium, suffer atrophy and absorption, partly in consequence of the pressure to which they are subjected by the growth of the connective tissue round them, and partly because their blood-supply is interrupted by progressive obliteration of the vessels concerned in their nutrition. At the same time it may happen that portions of the tubes may be constricted and their peripheral commencements suffer secondary dilatation, whereby *cysts* are formed. This connective tissue growth is very particularly liable to take place around Bowman's capsules, and leads to the obliteration of the Malpighian vascular tufts. The process does not extend equally in all directions, and hence one lights upon portions of the kidney, in a microscopical examination, which consist solely and entirely of fibrous tissue, while other parts of the same kidney furnish uriniferous tubules of a perfectly normal character with unaltered epithelial lining and healthy Malpighian bodies.

So far as this description of microscopical conditions found in contracted kidneys as given by the two English authors above mentioned is concerned, I could, according to my own observations and anatomical knowledge, heartily subscribe thereto, as well as to their ideas of the process whereby this state of things is brought about. But Gull and Sutton go further than this, as I have shown above, and consider this peculiar process that takes place round about the small vessels of the kidney to be, as a rule, merely one of the manifestations of a widely-spread general affection implicating the entire arterial system, or the greater portion of it. That this is correct I am at present far from being convinced. It is true that since their views on this subject were made known I have had no opportunity of testing their soundness upon the dead body; but I believe that from what I had previously observed and ascertained in the most unprejudiced manner with regard to genuine contracting kidney, and after all that Gull and Sutton have advanced concerning its etiology, I must express my entire dissent from this last idea of theirs.

This much, however, I believe to be beyond all doubt, namely, that *genuine renal contraction—the so-called third stage of Bright's disease* of our writers, *Grainger Stewart's cirrhosis of the kidney*—is the result of a primary growth or proliferation of the intertubular connective tissue, and commences and pursues its course quite independently of the other forms of renal inflammation previously described by me. It is, in fact, an altogether independent form of disease. *This process leads from its commencement steadily to the dwindling of the substance of the gland, a wasting preceded by no anterior inflammatory swelling of the organ.*

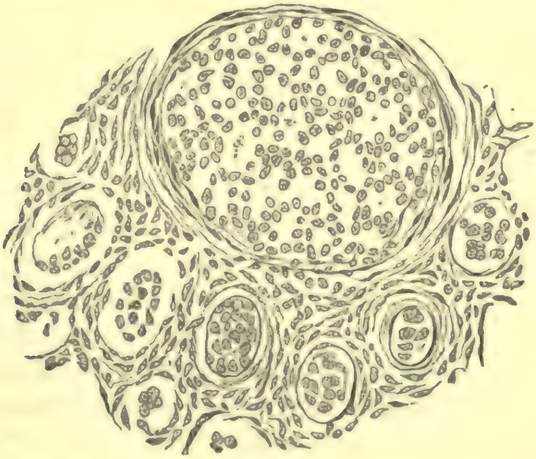


Fig. 13.

Cellular hyperplasia of the interstitial connective tissue. Section made from the cortical portion of a kidney in an advanced stage of contraction. (Colberg.)

The wasting here spoken of does not affect the whole mass of the cortical substance simultaneously, but commences in scattered spots upon the surface of the kidney and extends very gradually from the spots first implicated both upon the surface and into the depth of the organ.

These propositions are proved by the anatomical facts above adduced by me. I have never yet succeeded in tracing anatomically, in the dead body, the transition from inflammatory swelling of the kidney into that condition of true cirrhosis. As yet I have never met with a kidney one part of which was in a state of inflammatory swelling and another part perfectly normal, whereas I have frequently seen contraction of the larger portion of one kidney side by side with other smaller parts of the same organ that were not in the least implicated. Neither have others succeeded better than I, with cases that were watched clinically, in demonstrating such a career of the disease as would seem to indicate a probable consecution of conditions in the kidneys corresponding to those hitherto ordinarily accepted as constituting the three stages of Bright's disease.

From the description above given of the symptoms accompanying genuine contracting kidney and marking the course of this disease, the reasons will quite clearly appear which compelled me to dispute, from a clinical standpoint, the connection of this renal malady with the previously described inflammatory forms, and this conclusion was arrived at before I had been able to gain more precise ideas upon the actual anatomical process of the genuine contracting affection.

In saying this I do not pretend to deny that in *rare instances diffuse processes of inflammation may arise as complications in addition to already existing contraction of the organs.* I further consider it possible that *acute hemorrhagic nephritis may, although but rarely, give the start to the development of the contracting process.* I have myself communicated an instance above (Case XII.) whose course rendered some such connection of events as this probable.

Here I must guard my readers against confounding the *primary kidney contraction*, of which I am here speaking, with the *secondary form* previously adverted to, which is the occasional sequel of chronic parenchymatous nephritis. Johnson, Grainger Stewart, and more recently Gull and Sutton, have all prominently set forth the distinctions that obtain between them. In the first place, there is never such an excessive diminution of the kidneys arrived at in secondary atrophy as is observed, quite as the rule, in well-marked cases of genuine contracting kidneys. It may be considered quite an unusual event if a kidney which was once large and swollen again becomes reduced to the size of a normal kidney, and exceedingly rare for it to become smaller —Case XVII.

The surface of these kidneys is irregular and nodular, like the true contracting kidney; but the nodules are less uniform, many of them are considerably larger than are met with in the last-named form, and the nodosities are separated more widely from each other by broad weal-like bands of connective tissue, which adhere firmly to the capsule of the organ. These elevations upon the surface, as well as the patches seen in the substance of the gland upon section, consisting of recognizable remains of the glandular tissue, are distinguishable by their frequently intensely yellow color from the mostly dark brown color, or in very anæmic bodies, the whitish gray color, of the granulations which belong to the true contracting kidney.

Those little cysts in the cortical substance which are so frequently found in genuine contraction are far more rarely met with in secondary atrophy, and always occur in much smaller numbers. This difference was already made prominent by Grainger Stewart.

Microscopic examination shows that in the secondary contraction, comparatively speaking far larger numbers of the renal tubes are perhaps preserved in the cortical substance than happens in the primary disease, but that the epithelium is for the most part involved in fatty degeneration or reduced to mere fatty detritus; hence the yellow color of the remaining glandular substance. In the genuine contracting disease, on the other hand, the renal tubes still remaining are everywhere provided with quite normal epithelium.

Finally, Gull and Sutton failed to find any changes of the vessels in the secondary contracting disease. I cannot remember noticing, in the bands of connective tissue we meet with in the cirrhotic kidney, any of those fat globules deposited in lines, such as one so invariably finds distributed in the interstitial substance in secondary contraction, and which may be regarded as the remains of degenerated lymphoid cells.

Analysis of the Symptoms.

If we endeavor, now, from the above description of the symptoms of the disease and from the post-mortem appearances, to ascertain the physiological connection between the renal changes and those which take place in other organs, and strive to make out how far the disturbance of the renal functions depends upon the kidney disease, this peculiar circumstance must first strike our attention: that, despite the remarkable diminution in volume of the secreting structures, the quantity of the renal secretion exceeds, as a rule, its normal mean amount; and, furthermore, that the excretion of the specific constituents of the urine by means of the kidneys will often for years correspond to the mean physiological amounts. These and many other of the symptoms of genuine renal contraction will become clear and comprehensible to us only if we take into consideration the alterations in the arterial blood-pressure which the affection in question entails, almost without exception, and which are chiefly occasioned by the secondary changes which the heart experiences under the burden of the renal malady, viz., the hypertrophy of its left ventricle.

The almost constant coincidence of hypertrophy of the left ventricle with renal atrophy had not escaped Bright's notice. He sought its explanation in the irritation which he supposed impure blood, such as the diseased kidneys could not depurate of its excretory matters, must exert upon the cardiac muscular tissue. Bright, however, failed to prove that the blood was really already impure at the time when the heart-hypertrophy began. He failed to take into account the fact that the same source of irritation, whose existence he took for granted, and to whose influence he attributed the hypertrophy of the left ventricle, exerted no similar effect upon the right ventricle, through which the same blood, supposed to be thus impure, was flowing; and he overlooked the fact, that in other renal diseases where the blood was notoriously impure from the admixture of urinary constituents, no such hypertrophy of the left ventricle usually occurred.

These defects in Bright's doctrine did not escape Frerich's attention, and on their account he would not accept it, but inclined far more to the view that both the heart disease and the renal affection were co-results of one and the same evil influence, whether that were dram-drinking or exposure to cold.

Traube first insisted that the hypertrophy of the left ventricle was dependent, not upon the diffuse renal diseases in general, but upon that special variety or process of disease which led to contraction of the kidney. The proposition he put forward was that the hypertrophy of the left ventricle was the result of increased tension in the arterial system—a tension which must of necessity take place as soon as a great number of arterial branches in the kidneys, with the Malpighian tufts attached to them, were obliterated, and when the channels through which the blood of the renal artery must drain away were reduced within narrower limits. He placed the consequences of renal contraction, with perfect correctness, in the same category with the results which valvular deficiency of the mitral valve and certain diseases of the lung indisputably exert upon the right chamber of the heart.

Gull and Sutton again deny the connection between renal contraction and hypertrophy of the left ventricle as cause and effect. They hold both events to be joint results of the one general affection, which they claim is extended throughout the entire arterial system or spread over the greater part of it, namely, fibrous thickening of the coats of the vessels. In confirmation of their views they appeal to the circumstance that this heart-hypertrophy fails to appear in some instances of extreme renal contraction, and is absent, as a rule, in other kidney diseases; for example, scrofulous pyelitis, wherein the organs are well-nigh entirely destroyed. They will not admit Johnson's explanation of the non-appearance of cardiac hypertrophy in particular cases of renal contraction as due to conditions of general defective nutrition, because under these circumstances the kidney disease, if able under any circumstances to affect the heart, ought to have provoked dilatation of its cavities. Gull and Sutton's arguments, however, are really directed only against the doctrine defended by Johnson, that doctrine according to which blood rendered impure by defective renal secretion is the cause of the cardiac hypertrophy. They appear to be perfectly unaware of Traube's theory; at all events they make no mention of it.

I am unable to side with these opinions of the two English authors, partly because I am convinced that the premise on which they depend, to wit, the disease of the coats of the vessels pervading the entire arterial system or the greater part thereof, certainly does not hold good with regard to a large proportion of the cases of renal contraction, associated with hypertrophy of the heart; and partly because I am myself of Johnson's opinion that in states of general mal-nutrition, which include a watery condition of the blood or a general diminution in its mass, neither can any hypertrophy of the heart take place, nor need any dilatation of its cavities occur. If the blood be more watery than normal, as it is, for example, so regularly in chronic parenchymatous inflammation of the kidneys, there will be no abnormally high tension in the arterial system, or resistance offered calculated to increase the work of the ventricles, such as is the cause alike of hypertrophy and of the preceding or simultaneous dilatation of the heart, because transudation of the watery fluids into the cavities of the body and the subcutaneous cellular tissue prevents its occurrence. We learn this from clinical observation of parenchymatous nephritis in which the resistance offered to the blood's passage through the kidneys is often certainly no less than in advanced stages of renal contraction, as may be seen by the enormous tension of the arteries which occurs at the commencement of this affection; and still cardiac hypertrophy fails to present itself, because this elevated arterial pressure disappears directly upon the occurrence of dropsy and the failure of the general nutrition.

Again, if the general mass of the blood suffer diminution in volume, it is equally impossible for any abnormally high tension to take place in the aortic system, and here, too, hypertrophy of the left ventricle will not occur, even though both kidneys be atrophied. Indeed, we often find the right ventricle atrophied in cases of chronic pulmonary tuberculosis, which has obliterated larger areas of vessels within the lesser circulation than are ever destroyed in pulmonary emphysema, and yet the latter invariably leads to hypertrophy of the right ventricle. *The entire volume, then, and the character of the blood are factors which we must not leave out of our consideration in estimating the relation that subsists between atrophy of the kidneys and hypertrophy of the left ventricle.*

I am well aware that the absence of hypertrophy of the heart, where one kidney has suffered total destruction, can be adduced as evidence against cardiac hypertrophy depending upon obliteration of the renal vessels. Only here I throw out the suggestion that total wasting of one kidney is mostly accomplished under complications of exhausting disease, and that, before a favorable state of the general nutrition becomes re-established, the sound kidney has time to effect the well-known compensatory development of its substance, wherewith, no doubt, a considerable increase in its vascular economy is associated.[1] Up to the present time we possess no observations upon the extent or measure of the destruction of the blood-vessels that does take place in the cases of renal contraction that entail heart hypertrophy, and therefore do not know if the actual defect of blood-transit canals, by the destruction of the terminal branches of the renal arteries, in the well-marked cases of contraction of both kidneys, may not really be much more considerable than happens in case of the total destruction of a single kidney. Mere inspection with the naked eye certainly favors this view of the matter.

I am therefore compelled, from my acquaintance with the subject, and as the result of my observations, according to which the left ventricle of the heart, as a rule, likewise becomes hypertrophied in the secondary contraction of the kidney that follows parenchymatous nephritis, to express myself very distinctly in favor of the opinion entertained by Traube upon the mode of origin of cardiac hypertrophy in renal atrophy.

The extreme importance of this consecutive hypertrophy of the heart throughout the entire further pathology of the kidney disease here under consideration, must justify the length at which this subject has been discussed. The destruction of great numbers of blood-vessels in the kidney, on the one hand, and the hypertrophy of the left ventricle, on the other, may be regarded as the causes that determine the increase of the blood-pressure in the arterial system, and this is the key towards interpreting a whole series of symptoms which accompany the disease.

---

[1] Compare *Dr Leopold Perl*, Anatomische Studien über compensatorische Nierenhypertrophie. *Virchow's* Archiv. Bd. 56. p. 305.

The elevation of the arterial blood-pressure is of paramount importance towards understanding the secretory processes of the diseased kidneys. *Observation teaches us that contracting kidneys, which have dwindled down to more or less considerable remnants of secreting glandular tissue, do not merely continue to secrete urine, but in the large proportion of cases actually furnish, in the same interval of time, a larger quantity of urine than healthy kidneys would supply. This, however, takes place only so long as the condition of the hypertrophied left ventricle is capable of maintaining the blood-pressure in the aortic system at its abnormal height.* That the secretory performances of the kidneys depend upon the elevation of the pressure in the arterial system, is proved as distinctly as is possible by physiological experimentation. If the arterial pressure exceeds its normal bounds, it follows of necessity that, *cæteris paribus*, a larger quantity of urinary fluid must be forced through the renal filtering apparatus during the same interval of time than would take place under normal pressure. Even in this way—at least so we can imagine—the diminution in secreting surface caused by the process of contraction can be covered, or even more than compensated for. Another circumstance which must not pass unheeded is that the degree of hypertrophy of the heart, and therefore its compensating powers or capacity, will vary in different cases in accordance with the general state of the patient's nutrition and the external conditions to which he is subjected, and that, as a matter of course, the same factors will materially influence the course and issue of individual cases of the disease.

The increase of the arterial blood-pressure does not, however, cause a larger secretion of urine than normal, merely because more fluid is forced into the kidneys through what is left of effective filters; this fluid is also urged with greater rapidity through the uriniferous tubules by the pressure of the filtrate from behind it, and is, therefore, during its transit to the urinary passages, subject to less alteration by endosmotic and exosmotic currents. It will not, therefore, lose the greater part of its water by diffusing this into the concentrated blood-serum that lies in the capillaries surrounding the uriniferous tubules, as takes place under conditions of slower secretion, under normal blood-pressures; and it will absorb less solid constituents from the blood-serum, and take up into itself less rinsings from the epithelium of the uriniferous tubules. For experiment has shown us that to effect this a certain lapse of time is requisite; the less, therefore, the rapidity with which secretion takes place, the more concentrated becomes the secretion. Polyuria, the ordinary symptom of renal contraction, is, therefore, a result of increased blood-pressure in the aortic system.

The greater rapidity with which the secretion of the urine is conducted is, at the same time, the cause of its possessing so invariably low a specific gravity, i. e., of its remaining so relatively poor in solid constituents.

But, as soon as the propulsive power of the hypertrophied heart is reduced in consequence either of some temporary or permanent influence, the abnormally large amount of urine falls off, and the abnormally low specific gravity rises. Indeed, under such circumstances, the diurnal quantity of urine secreted may sink below its ordinary physiological mean, as tables above given prove,—the best possible evidence that the secreting function of the kidneys depends upon the blood-pressure. Corresponding with the diminution in amount of the urine secreted, under the conditions we are supposing, it is true that its specific gravity will rise; still, it always remains below its physiological average, unless some quite abnormal influence comes into play, like the disturbance of the circulation which is associated with epileptiform convulsions.

Generally speaking, however, we may agree entirely with our excellent investigator Traube, who regards *the hypertrophy of the left ventricle as an efficient compensation for the loss of renal secreting tissue*, such as the kidney contraction carries with it. This holds good, too, not only for the excretion of water, but for that of the urinary solids as well.

The renal secretion, which, as a rule, is excessive in quantity, is, in truth, comparatively poor in solid constituents, especially in urea. It seems natural to assume that the destruction of so large an amount of gland-cells, which, as no one will deny, possess special influence in excreting the specific urinary constituents, may be held responsible for a reduction in the amount of urea in the urine furnished by contracting kidneys. But if we test the total performances of these diseased kidneys, in this particular respect, by careful analysis of the urine, we shall find, what the results of my own analyses adduced above evince, that the small percentage of urea and salts in this urine is made up for by the large amount of urine passed, and this to such an extent that the diseased kidneys, under certain circumstances, may be in a position to excrete even more urea than healthy organs are in the habit of doing. Thus, one of my patients, as the mean of seventy-six analyses conducted over a period of six months, daily excreted over thirty-three grammes of urea, and upon one day as much as fifty grammes. Nature, in this as in so many other respects, has constructed the human organism with a very lavish hand, allowing it a great excess of renal parenchyma.

Under ordinary circumstances the kidneys need not work up to the full measure of their capacities in removing the superfluous water and purifying the blood from those remains of tissue metamorphosis which it is intended the kidney should excrete. This fact is already evident from the well-known circumstance that there are men in whom one kidney has been destroyed while still in an embryonal state, who have still developed perfectly naturally with the help of only one kidney, and have grown and thriven, living long lives, and fulfilling every duty of their lives fitly ; no single circumstance about their urinary functions revealing the defect in them of one entire half of the urine-secreting apparatus. But a grown-up man may, even after losing a kidney, by the help of the one he possesses, perfectly satisfy the secretory requirements of his economy, and once more regain as well as maintain a perfect state of health. This was shown me by a robust sailor who came under my observation, and died in my wards of small-pox in 1873—a man from whom B. Langenbeck had removed a vesical calculus in 1847 by the high operation. In his body we found, in the place of the left kidney, only a small lump of cicatricial connective tissue, which contained scarcely a remnant of renal tissue in it. In the contracted recess of the pelvis there was an oxalate of lime concretion. The thing, however, is illustrated in the most striking manner by the well-known case of our gifted surgeon Simon, who ventured to extirpate a kidney, and who had the good luck to keep alive the first person on whom this hazardous feat was performed.

The fact that, even in high grades of renal contraction, all symptoms which depend upon the retention of nitrogenous excreta in the blood and tissues may in some cases be absent, appears to me to be explicable only on the theory, adopted by myself and rendered so probable by the experiments of Heidenhain, to the effect that the renal epithelium takes part in the excretion of the specific constituents of the urine. If we suppose that the attraction of the epithelium for urea or its preliminary chemical compound circulating in the blood depends upon the amount of this material conveyed to the cells which are still active,—and the analogy of all other secreting glands favors this view,—then the renal epithelium of the contracting kidney, by reason of the rapidity with which the blood flows to it, will certainly be in a position to attract a larger quantity of this substance towards itself than the same cells in sound organs under conditions of normal blood-pressure could take up. Under these circumstances, the epithelial cells which remained active in a contracting kidney would tend to become overloaded with excretory materials, and either an arrest of the secreting function would most likely ensue, or the urinary elements already secreted would be reabsorbed into the blood (an event well known to follow upon obstructed outflow to the biliary secretion), if the substance thus secreted were not being continually washed out of the epithelial cells by an abundant current of water. This, however, is what takes place, and thus the functional capacity of the cells is restored, and they are enabled to attract fresh urinary constituents from the blood towards themselves; and, in point of fact, in genuine contracting renal disease, this process is continued for a very long period of time.

So long as the hypertrophied heart labors energetically, no uræmic symptoms occur, for an over-abundant secretion of urine is being carried on, and in consequence there is sufficient rinsing and washing-out of the secreting gland-cells. But, directly this excess of blood-pressure fails, from any cause, or the urinary secretion, in consequence of transitory or permanent weakness of the heart, is diminished, uræmic symptoms set in, and not rarely dropsy sets in at the same time, or even before. *Still, the inadequacy of the remnant of renal substance, for its task of excreting the specific urinary constituents and for the task of excreting the excess of water from the blood, does not, by any means, always cover the same period of time. Dropsy may precede uræmia or uræmia dropsy, and either symptom may disappear again without the other necessarily ensuing.* This may happen, too, long before the wasting of the renal substance has advanced so far that what remains is absolutely insufficient to fulfil its allotted functions. We learn this from the cases where patients survive their first acute uræmic attack for years.

But, besides the polyuria and the low specific gravity of the urine in contracting kidneys, there is another peculiarity in this disease which is explicable on the ground of this abnormally high arterial pressure, namely, the *albuminuria*. In genuine contracting kidney, the renal structural changes within the kidney are not in and of themselves the cause of the albuminuria, as is still largely taught in England; *the albuminuria is entirely due to the increase of the blood-pressure in the vascular tufts.* There is no better proof of the correctness of this proposition than is proffered by the exceptional cases. Thus, in our Clinic, as already stated, four cases of genuine contracting disease came under observation in which the affection was not far advanced, and its existence was only inferred from the albumen contained in urine which was secreted in pretty fair quantity, and from the presence of a demonstrable simple hypertrophy of the left ventricle. In each one of these cases it was entirely in our power to make albumen appear or disappear from the renal secretion at will. As soon as the patients were confined to their beds, and the rest thus enforced reduced the sum total of the heart's pumping force, and at the same time diminished the general arterial tension, the urine ceased to contain albumen. Whereas, so soon as the patients, who were still perfectly vigorous, went about in the open air at pleasure, albumen at once reappeared in the renal secretion (compare Cases XXIV. and XXV.).

In what has been advanced I believe that I have referred the peculiar conditions of the urinary secretion, such as we encounter clinically in the genuine renal contracting process, to their physiological causes. But it still remains for me to show the connection and dependence which exist between several other symptoms which these patients present, and the state of the secretory functions of the kidney above spoken of, and to establish the difference between these manifestations and those encountered in other kidney diseases.

In the first place it is manifest that the functional disturbance of the kidneys in the progress of contracting disease is not calculated at its commencement to alter the constituents of the blood materially, and therefore, especially as long as the polyuria so characteristic of this disease continues, no hydræmia will occur. Hence, it comes to pass that this affection, in more than one-half of all the cases, terminates fatally without the patient ever having been dropsical. Unfortunately, I possess no analysis of the blood, instituted in the early stages of the malady, that would directly prove the correctness of my assertion. The facts, however, which I have just dwelt upon speak with sufficient weight and emphasis in my behalf.

The excellent state of nutrition of the body is evidence no less valuable in proof of the natural quality of the blood during a considerable period of the course of the disease. In some instances the strength and activity of the persons affected is for years not notably influenced, and yet, as a rule, the renal affection is already distinctly recognizable when the first symptoms of the disease appear, and it is impossible to say how long it may have already lasted. The usually small loss of albumen in the urine affects the general nutrition so very little, while the organs of digestion continue to fulfil their functions regularly, that its influence is scarcely to be remarked, although it may have lasted perhaps for years.

But before the destruction of the kidney substance has attained any extreme degree, and the kidneys have in this way become absolutely insufficient for excreting the excess of water and excrementitious matters from the blood, some particular circumstance may induce such insufficiency. This premature insufficiency of the kidney may involve one or the other, or else both of its above-mentioned physiological tasks; for, as stated above, the inadequacy of the affected organs to perform their secretory functions does not always manifest itself simultaneously in both directions.

Dropsy occurs, so to speak, prematurely, when the propulsive force of the heart is diminished by any weakening cause, as for example in consequence of scanty subsistence, from the deteriorating effects of drunkenness upon the heart's muscular substance, or from disease of its valvular apparatus. Any other mechanical cause that disturbs the circulation (like considerable pleuritic exudation) will act in a similar manner by diminishing the increased arterial blood-pressure habitual to renal contraction. Occurrences of this kind are always followed by a very considerable reduction of the urinary secretion, which, as shown by the examples above given, may fall far below its mean physiological amount. If the diminution be permanent, dropsy will set in; but uræmia need not necessarily occur at once, for the same cause which prompts the failure of the heart's action often lowers the metamorphosis of tissue, and is therefore associated with diminished production of nitrogenous excrementitious matter. Nevertheless, on account of the diminished rapidity with which secretion is taking place, the more concentrated urine contains relatively more specific constituents than it did before. Finally, a sufficient amount of nitrogenous excrementitious matter to be worthy of mention leaves the circulatory system in company with the dropsical effusions. Thus it is that what still remains of the renal tissue suffices even yet to fulfil its depurating task, although the lessened arterial pressure is no longer able to excrete water enough by the kidneys to obviate hydræmia and dropsy.

It stands to reason that in renal contraction which is but little advanced, dropsy of this kind may completely and permanently subside again, if the concurrent causes which determined it are removed, and the blood-pressure throughout the aortic system rises to its previously high tension.

More frequent even than such premature dropsy, in the earlier periods of this disease, is a condition of things in which the affected kidneys are inadequate to the task of purifying the organism from its nitrogenous waste material, and thus severe uræmic symptoms ensue. In such cases as these I have not succeeded in finding out the actual grounds of this temporary disproportion between the production of this excretory material in the body and its excretion by the kidneys, for it is to some such disproportion that we must attribute the provocation of these severer symptoms. For in such cases where uræmic symptoms break out prematurely, we have usually no opportunity of investigating the secretory capacity of the kidneys before the uræmia sets in; attacks of this nature ordinarily arising in individuals who previously considered themselves in health. If such individuals die of their first attack, we find their kidneys only moderately altered, and may conclude from this circumstance alone that the organs were, under ordinary conditions, in a position to satisfy the demands which the organism made upon their secreting powers. Similar examples, however, in which the patients recover from grave uræmic fits, and continue to live on, feeling tolerably well for a long time, and even pursue their avocations again, prove in the most striking manner that *temporary circumstances, and such, too, as in all probability lie outside of the diseased kidneys, are capable of determining a transitory inadequacy in these organs for fulfilling their depurating functions.* Such cases have repeatedly come before me.

Case XXVI.—Herr B., the literary gentleman whom I have previously mentioned, aged fifty-seven years, had some years before recovered from paralysis of the extensor muscles of both his forearms, consequent upon lead-poisoning—the lead having been absorbed from snuff. In the summer of 1867 he had suffered with severe attacks of migraine oftener than had been his wont of late years—attacks which he attributed to the incessant worry which his official situation involved. Having come home from a meeting where there had been some violent scenes, he was seized during the afternoon of the 6th of December with an epileptic attack. His tongue was bitten in this fit, and it was succeeded by prolonged coma, from which the patient only quite recovered in the course of the following day. His urine, examined after the attack, showed all the characters which ordinarily appertain to this secretion in contracting kidneys: it was pale, of low specific gravity, containing both albumen and casts, but was passed in only small quantity (880 c.c. between the 7th and 8th of December). Later on the secretion became abundant, but the urine continued albuminous.

After this attack Herr B. for some months still continued to administer his arduous office, but died on the 25th April of the following year of cerebral apoplexy, which assailed him in the midst of a lively entertainment in a friend's house. The *post-mortem* showed considerable contraction of both kidneys, with numberless cysts in the scant remains of cortical substance. Enormous hypertrophy of the left ventricle of the heart, valves normal. Fresh apoplectic clot of the size of a fowl's egg in the left corpus striatum and its immediate neighborhood, which had burst through into the left lateral ventricle, the blood having made its way thence into both the third and the fourth ventricles, as well as into the right lateral ventricle. Another older apoplectic cyst, about the size of a bean, was found in the left thalamus. No trace of dropsy.

Case XXVII.—H. H., thirty years of age, a vagabond drunkard, was first admitted into my clinic in October, 1866, on account of dropsy, and the diagnosis of Bright's disease was set against his name in the patients' ward book. No minute history was recorded. His second admission was on November 10, 1872. The previous night the patient had had repeated epileptiform attacks; when admitted he was completely insensible, his body was slightly swollen with anasarca, and his skin, especially that of the abdomen, had been much scratched. The temperature in the rectum was 40.6° C. (105.1° F.); the pulse was frequent, regular, but feeble. There was distinct hypertrophy of the left ventricle. Directly after his arrival at midday he had a fresh attack, and altogether had twelve more fits up to two o'clock in the night. In some of his blood, drawn by venesection and analyzed by Prof. Jacobson, one-tenth of one per cent. of urea was found, but no ammonia. On the 11th of November the patient passed plenty of water in his bed, was sensible, but sleepy. Temperature normal. Pulse quiet. Anasarca nearly disappeared. After this the fellow had an attack of delirium tremens, and the diagnosis of contracted kidney was established upon examination of his urine. He was allowed to go out at his own wish on December 10th, and was seen lying dead drunk in the gutter the very same evening.

On the 18th of December he was once more admitted into the hospital; he was very slightly œdematous and his urine contained some blood. On the 28th of January, 1873, he left the hospital feeling well.

On the evening of July 27th he was brought back again to us on account of total blindness, which had come on quite suddenly. Ophthalmoscopic examination threw no light upon its causation, and after a few hours the sight completely returned again. Still his legs, and more particularly his face, were highly œdematous. Urine was passed in abundant quantity, and was pale and albuminous, as it had been before. Under good care and the use of hot baths he this time again recovered quickly, and this so completely that in September he was able to undertake the care of some cholera patients who were placed in a separate barrack building. Here day and night he attended to his charge with the greatest fidelity, and exerted himself to his very utmost. At the end of September he felt unwell, and on October 1st I found him the victim of a well-marked attack of cholera. On the evening of October 3d he died, after passing four entire days without secreting any urine and lying in a prolonged state of coma.

Instead of a full account of all that was found at the *post-mortem*, I give here only the diagnosis made upon it by my colleague Heller: *extreme granular atrophy of both kidneys, considerable hypertrophy of the left ventricle, etc., etc.*

I have hitherto repeatedly ascribed a compensatory action to the hypertrophy of the left ventricle, my purpose being to show that, in consequence of the greater activity of the heart's power, the secreting tissue that still remained in a contracting kidney was enabled satisfactorily to accomplish its secretory task ; but I must now refer to the dangers and disadvantages with which the increased arterial tension produced by this hypertrophied heart threaten the patient. The evil influence of the condition of things we are considering is exhibited in its simplest manner by the relatively frequent occurrence of cerebral hemorrhages in genuine contracting renal disease, the true explanation of which no one, surely, will seek elsewhere than in the notoriously abnormal high pressure maintained in the arteries, inasmuch as cerebral apoplexy in renal disease may attack persons who are comparatively young, and whose arterial walls are quite healthy, as illustrated by Case XXIV.

I have already stated that the dangerous hemorrhages from mucous membranes, of which several cases above cited offer instances, ought not, in my opinion, to be attributed to the increased pressure in the arterial system. They may well own a similar pathology to the perilous and often fatal bleedings that take place in cirrhosis of the liver and in leukæmia, etc., and be due, perhaps, to abnormal states of nutrition in the coats of the vessels.

On the other hand, I do not hesitate to attribute the attacks of palpitation, distress, and vertigo, of which some of our patients complain, to the increased arterial pressure.

Since reading Gull and Sutton's memoir, however, I cannot help suspecting that the pains in the head which torment our patients often for years before their deaths may be due to the same cause. In a great many cases of this nature I have, as above noticed, found the skull-cap after death extraordinarily thick and compact, and the *membranes, both the dura and the pia mater, also greatly thickened.* It is obvious that the process by which this abnormal condition of the parts above mentioned is brought about must or might be attended by the sensation of pain. I have put the question to myself whether the idea of the two English authors could be correct, which attributes the thickening of the membranes of the brain to a separate process quite independent of the renal disease, albeit analogous to it, but at present must leave this as a mooted point. The above-named English writers regard retinitis Brightica in exactly the same light as a spontaneous process of disease independent of the renal affection, and one merely frequently occurring at the same time. For the following account of this branch of the pathology of the renal affection in question I am indebted to the goodness of my friend and colleague, Voelckers. It appears from Voelckers' experience that Gull and Sutton's conception of the process is incorrect.

Voelckers: On Retinitis Albuminurica.

"It has long been known that disturbances of vision took place in renal disease, but the causes of this mysterious amblyopia could only be more closely investigated after the discovery of the ophthalmoscope. We know now that these disturbances of vision own a twofold origin, the one cerebral, designated as amaurosis uræmica, and induced by the surcharging of the blood with excretory materials retained in it; the other local, provoked by structural alterations in the retina, and called retinitis albuminurica. Whereas in amaurosis uræmica the disorder of vision commences suddenly and may as suddenly subside again, being usually accompanied by other symptoms due to the fundamental malady, we encounter obscurities of vision (amblyopiæ) in chronic nephritis, which, commencing quite gradually, show themselves, as a rule, as simple diminutions in the acuteness of the sight.

"The diminution in the sharpness of vision develops itself so gradually, and, in spite of well-marked changes in the retina, may be so slight, that the patient is hardly aware of it himself. Hence, it happens often enough that the ophthalmoscopic examination gives the first information of the grave general affection that exists. Sometimes the patients complain of temporary deterioration in their sight, of momentary darkening of the field of vision which overtakes them principally during excitement or when they are exerting themselves. In nervous, excitable patients I have repeatedly been able to make out a difference in the visual powers during one and the same examination. In some cases one discovers distortions of vision (metamorphopsia)—crooked sight—(*i. e.*, straight lines seem bent.—Translator). During the commencing stage, ophthalmoscopic examination shows a slight, and for the most part striped cloudiness of the papilla of the optic nerve and of its immediate neighborhood. It looks grayish red, is swollen, its margin is indistinct and blurred. One of the most striking features is the state of the veins, which are enlarged and tortuous, and exhibit differences of color, according as they run, in their serpentine course, now upon the surface, and now deeper in the substance of the retina; the shade of color being always darker the nearer the surface the vein happens to lie. Beyond occasionally presenting a constricted calibre, the arteries show no abnormity. The retina surrounding the optic nerve is likewise cloudy, as already stated, and exhibits extravasations of blood which are more or less numerous, conceal the vessels in places, and are either striped, round, or irregular in shape. Besides these, we perceive white spots of very various forms, from the finest stippled points up to the larger patches, as large as the diameter of the papilla itself, or even larger. Even at this stage we discover the changes that are most characteristic, if we get the patient to look straight into the hole of the mirror, thus bringing the yellow spot into view. Its central portion is generally unaltered, but in its periphery, and especially in its outer or temporal side, one sees fine white dottings and radiating lines, which give the impression of having been splashed on with a feather.

"In the course of time the cloudiness of the retinal tissue increases, the grayish white exudations grow larger, run together, and surround the intensely swollen papilla with a thick rampart, broader than the diameters of several papillæ put together, and sending out projections which mostly follow the course of the larger vessels and sometimes obscure them. Larger and smaller extravasations of blood stand out with singular sharpness against the light background; the streaks and stipplings over the macula lutea become more and more confluent, so that the mass of exudation that surrounds the optic nerve extends continuously over its transverse diameter. Further toward the periphery, in the equatorial region, the background of the eye appears to be normal, aside from a distinctly visible amount of venous stasis. This picture of retinitis, carried to its fullest development, is certainly but rarely seen. As a rule, the exudations are much less considerable; frequently enough the cloudiness of the papilla and of its neighborhood is but little marked, and only a few plaques (patches) and a few scattered blood-spots, besides the dottings of the macula lutea which are rarely absent, arouse our suspicion of the grave renal disease.

"The changes are quite generally distinctly marked in both eyes, although it may be to a very different extent. In three cases I recognized the affection in individuals sent me by my colleagues with the diagnosis of embolism of the arteria centralis retinæ; in each instance I was able to confirm this diagnosis. I pass by the question whether this embolism stood in causal relationship with the renal affection. In no one of these cases was there any valvular lesion of the heart; in one there was well-marked hypertrophy of the left ventricle. But in addition to this, in two cases especially, I remember that the first signs of commencing retinitis albuminurica were presented in the eye that was presumed to be healthy. It presents such typical features that the ophthalmic physician, even while no physical sign of a general malady is present, is justified in inferring the existence of renal disease from the ophthalmoscopic appearances alone.

"Numerous observations prove that the retinal changes above described, even though they have attained a considerable extent, may subside again. The white patches or areas become smaller from their periphery, they break up by becoming cleft, the vessels that were originally concealed by them become visible once more, and at last even the striped cloudiness of the retina disappears, the margin of the papilla becomes sharper in its outline, and often nothing further than some thickening of the walls of the vessels remains to be seen.

"Complete reabsorption, however, certainly occurs only in that form of retinitis which accompanies the acute nephritis of pregnancy and scarlet fever, and in these cases vision may become completely restored. Of two cases of retinitis albuminurica in pregnant women which I have seen, one obtained perfect eyesight again ; in the other case vision improved, but albuminuria with retinitis recurred in an ensuing pregnancy, when extensive atrophy of the optic nerve took place followed by absolute amaurosis.

"The retinitis attending chronic nephritis certainly admits of improvement ; at times one sees the sight increase, and now and again he notices some diminution in the retinal changes, but recovery or even permanent amendment is probably never met with.

"Retinitis albuminurica occurs in every form of renal inflammation ; several observers, too, have found it in amyloid degeneration ; out of fully thirty cases observed by me, two were noted as being incident to pregnancy, all the rest happened in individuals in whom the diagnosis of *granular atrophy* of the kidneys had to be made.

"As a matter of course the treatment coincides with that of the general malady. I have only seen temporary amendment result from the blood depletions which have received such high commendation ; in the long run I believe they do more harm than good. I think that I have found these patients to do best on good strong nourishment and the use of the iodide of iron."

So soon as the destruction of renal tissue has advanced to such a stage that the remains of the glands are no longer adequate, although assisted by what may be called the compensatory elevation of the arterial blood-pressure, to fulfil the task of secretion demanded of them by the organism, then the results of this insufficiency never fail to demonstrate themselves. Dropsy, chronic uræmia, or inflammatory affections of the tissues, fanned into a flame by the chemical effect of the excrementitious materials accumulated in the blood, present themselves as terminal symptoms, being generally preceded by marked loss of strength and evident marasmus, due to digestive disorders, vomiting, diarrhœa, or hemorrhage. It is these last-named circumstances principally, which, by reaction on the general nutrition, induce degeneration of the hypertrophied heart-muscle, and hence reduce the aortic arterial pressure, thus upsetting the arrangements calculated to compensate for the loss of renal tissue. In such cases, months even before death, the pulse, which had been tense, full, and elastic, becomes small and soft—the heart's impulse, lately so heaving, is scarcely perceptible, the sounds are no longer loud, and only the second sound over the aorta preserves its distinct accentuation.

The Duration of Genuine Contracting Atrophy of the Kidneys.

It is clearly impossible to say anything definite with regard to the duration of a malady whose commencement nearly always completely escapes detection. If the disease has advanced far enough to be detected with certainty, it is quite sure already to have existed for some length of time, for the changes which mark its existence, as for example, the hypertrophy of the left ventricle, require some considerable time for their accomplishment, and follow only upon a process of disease which is of slow development. I have already said that I have some grounds for thinking that the cases of renal disease which occur in the course of acute articular rheumatism may lead to contraction of the kidneys. Unfortunately, both my own cases of this nature were complicated by valvular disease of the heart, thus introducing an element of obscurity into the observation. Instances of renal disease arising in connection with uncomplicated cases of acute articular rheumatism might, accordingly, if my supposition should prove to be correct, afford the best opportunity of determining the precise beginning of the disease, and consequently its duration in any individual case.

This much, however, is established with certainty by the foregoing observations, that the affection, as a rule, is a very protracted one, extending in a great many instances over several years, or, at all events, being capable of lasting so long as this, if not interrupted prematurely by some of the frequent complications which induce sudden death. We cannot at present determine whether external circumstances have any influence in accelerating the development of the renal disorder, or, if so, what particular circumstances have this power. But, on the other hand, it can be demonstrated with great certainty that all circumstances which materially weaken the heart's action imperil the life of the patient, before the destruction of renal tissue has reached that extreme grade which may be observed in other patients, the strength of whose hearts has remained undiminished, and who remain in a tolerably comfortable condition until suddenly carried off by apoplexy or uræmia. When I collect all my own cases in which the diagnosis has been confirmed by an autopsy, I find that the longest period, which elapsed between the date when the affection was first ascertained with certainty and that of death, was observed in the case of the drunkard, H. H.; it embraced a term of seven years, and even then he did not die of his renal disease, but of cholera. I watched the disease in the brewer K. through a period of three years, he being, also, repeatedly in my Clinic and under my care. During this time his general condition varied considerably with the sort of life he led. While in the hospital and well cared for he did well, increasing in weight and strength, and having nothing to complain of; outside the hospital he lacked nutritious and sufficient food, took to drinking, and after a while returned to our care, pale, feeble, and dropsical, again quickly to recover under appropriate treatment. At last he was shut up in the poor-house, where, after the first symptoms of chronic uræmia, vomiting, and a certain amount of delirium had set in, he put an end to the matter by destroying himself.

Prognosis.

Our present experience of genuine contracting kidney compels us unfortunately to accept Grainger Stewart's dictum, that "this is the most hopeless of all the forms of Bright's disease." Thus far I have heard no competent authority state that he reckoned the disease curable. No one would be prepared to assert that kidney structures once destroyed, be the cause of their destruction what it may, ever could be repaired again—or, in other words, that a *restitutio in integrum* was feasible.

But it is another question whether an arrest of the process, once established, may not be possible, constituting a sort of comparative recovery. But even here such experience as we possess is not encouraging. Among the very large number of bodies opened in our post-mortem rooms we should, in that event, necessarily often alight upon partially atrophied kidneys. It is true that no one could tell from its anatomical aspect what further shrinking the diseased kidney might have undergone, if sufficient time had been allowed; but the symptoms, if they are observed during life, inform us that the function of these atrophied organs is at least not regularly fulfilled. I cannot as yet give a decided negative answer to the question whether medical skill is in a position to arrest the destructive progress of the disease if this is recognized early. Indeed, the question itself might be considered a vain one so long as contraction of the kidney was reckoned in all cases to be the final issue of an inflammatory process. But, since it may be held established that the process of contraction represents a process of disease which runs its course independently, we must admit that the absolutely bad prognosis which is universally given in these cases is the result of our preconceived ideas or judgment.

For myself, it is true, I cannot say positively that I ever saw a case of genuine contracting disease arrested in its progress, thus resulting in a comparative cure; still I would not by any means dispute the possibility of such a result being arrived at by therapeutic means, if the disease were only recognized sufficiently early. The observations we at present possess upon the earliest stages of the malady, not to say its absolute commencement, are so few and so defective that the matter is by no means ripe for decision.

I unhesitatingly announce it as my opinion that greater care in the examination of patients will lead to an earlier recognition of this insidious malady, and that the knowledge of its grave import will conduce to its more attentive handling. But not until physicians accord that attention which it deserves to the condition of the kidneys of their patients shall we succeed more frequently in catching the process of contraction at its very outset, and not till then shall we be able to determine the power of therapeutic measures to control the progress of this dire disease.

But if the affection, as is now generally the case, is not recognized until large portions, or perhaps the greater part, of the kidney is irreparably lost, and when dropsy or uræmia testifies to the actual insufficiency of what remains, then indeed the sentence of death which is universally pronounced upon any one affected with contracting kidney may be justified; and then the prognosis, in any particular case, will only occupy itself with the question of the immediate degree of danger in which the patient is placed. The degree of functional activity of the kidneys and the condition of the heart must be the guides to the diagnosis in each individual case. But I have already adduced what I had to say on this subject above, and especially dwelt upon the significance that attaches to the increased labor of the hypertrophied heart.

Next to persistent reduction in the quantity of the urine and simultaneous diminution in the amount of its solid urinary constituents, obstinate vomiting or diarrhœa are the surest harbingers of speedy decease.

Death is indisputably close at hand when such *symptoms of chronic uræmia* as *itching of the skin* and *drowsiness* set in; and equally near is it when *hemorrhages take place from various mucous membranes. Acute uræmic attacks* of the severest kind may pass over without *immediate* death necessarily ensuing. *Dropsical swellings* may disappear, and life be prolonged for years without their recurrence.

The affection of the retina is very often established long before death, and while the patient otherwise feels perfectly well; still, most of my patients had already become anæmic and lowered in their state of general nutrition when their eyes became affected.

After *cerebral apoplexy*, and even after this has induced insensibility of several days' duration, I have still seen remarkable recoveries. The brewer K., whose case was given as No. XXIV., after an attack of apoplexy, suffered with hemiplegia and aphasia. But these symptoms had almost entirely subsided six months later, when he died of hemorrhage from the bowels.

The intercurrent *inflammations of the serous membranes, erysipelas, carbuncle*, and *pneumonia*, nearly always have a fatal issue. These complications are far more dangerous, according to my experience, in contracting kidney than in parenchymatous nephritis. A good state of general nutrition and strength justifies the hope for a prolongation of life; should the nutrition fail and the strength visibly decline, and a pale, ashy hue pervade the general surface of the skin, a speedy fatal end may be anticipated.

I never yet saw a single patient of this kind die of marasmus alone. Death was nearly always ushered in by symptoms of chronic uræmia, less frequently by inflammatory processes in other organs, and sometimes by uncontrollable hemorrhages from some mucous surface.

Diagnosis.

I have repeatedly laid stress upon the circumstance that genuine contracting kidney develops itself so insidiously that the malady, at its commencement, is attended by no striking symptoms. Even the first signs of the disease are of such an indefinite nature, and may be so different in the different cases, that they cannot be designated as in any respect characteristic, and in no instance do they point directly to the kidney as the seat of disease. He only who makes it his rule to probe to its utmost every symptom of disease, with the intention of discovering its possible cause, and who *examines the urine of his patients whenever they persist in remaining poorly—he only will succeed in recognizing this insidious renal malady betimes.* Headache and vertigo, of a manifestly congestive nature, attacks of palpitation and difficult breathing, at once direct attention to the heart. Then if, on physical examination, the organ is found enlarged or its impulse excessive, if auscultation shows its sounds to be loud and clear, indicating that the valves are normal, and especially if the diastolic sound over the aorta is greatly increased in strength, and the pulse is unusually tense, the diagnosis of renal contraction becomes highly probable. But it can only be established with certainty by the examination of the urine.

The large quantity of urine secreted daily, its pale color, its low specific gravity, the small amount of albumen it contains—all these are tokens which, placed beside a hypertrophied heart, render the diagnosis of kidney contraction positive. But I have already remarked in a former chapter that contraction of the kidney may follow as a secondary process after diffuse nephritis, that this form of renal contraction may also induce hypertrophy of the heart, and that under these circumstances the urine may in reality assume essentially the self-same characteristics. Too few of these cases have come before me in my hospital practice for me to lay down with certainty any distinguishing features of the urine which might serve to establish the differential diagnosis. Still, I would remind my reader of the fact stated above, that, according to my hospital experience (my observations extend to only two cases, in which, after general dropsy had lasted for years, apparent recovery ensued, but the albuminuria still persisted for years), the daily average amount of urine is below the ordinary quantity furnished in genuine contracting disease; furthermore, that the urine in secondary contracting kidney, as a rule, deposits a sediment forming a considerable, pale gray, dust-like stratum, which collects towards the bottom of the vessel, and consists of an astonishing amount of casts, which are chiefly thick casts, some of them highly granular and opaque, but some of them also yellow-colored and highly refracting, as well as of large quantities of granular detritus. Numerous fragments of degenerated epithelium stick to the casts. On the other hand, the urine furnished by kidneys in a state of genuine contraction nearly always presents very few casts, and these are chiefly of the perfectly hyaline variety, and nearly all of small size. Then, if an epithelial cell is seen attached to any such cast, it will be, as a rule, perfectly normal in its appearance. But in any doubtful case of this nature the diagnosis may be assured by the help of the history. Albuminuria occurring in an individual *who has been very dropsical for a considerable period of time*, could hardly be induced by genuine contracting kidney.

But I must again utter my warning against being content to establish a diagnosis upon the examination of a single specimen of urine. I have already, in speaking of nephritis, put forward the fact that inflamed kidneys will at times secrete large quantities of urine of pale color and with a low specific gravity. Here, it is true, one finds large numbers of casts, but casts most of them very different in character from those found in the urine furnished by cirrhotic kidneys. Exceptions to this rule, however, will occur. Still, for these cases the circumstance is certainly worthy of note, that, as a rule, a large amount of colorless blood-cells appear in the urine from inflamed kidneys, while these are entirely absent in that supplied by cirrhosed kidneys. Here, however, there is one possible source of error, for in the latter instance it happens not very rarely that cytoid (mucus) corpuscles become mixed with the urine, being derived from a catarrhal condition of the urinary passages.

The secretion furnished by kidneys involved in *amyloid degeneration* may exhibit entirely the same characteristics as the urine of cirrhotic kidneys. In amyloid disease the urine is not rarely passed in excessive quantity, and then is just as pale and of as low specific gravity as the fluid derived from genuine contracting disease of the kidneys; it may contain, too, just as small an amount of albumen, and even fewer casts (occasionally, after a prolonged search, we find none at all); but, if any are present, they are nearly always of the small variety, and as invariably of perfectly hyaline character. Should epithelial cells be seen attached to these casts, they, too, like those seen adhering to the casts from cirrhotic kidneys, will be perfectly normal in appearance.

The distinction, therefore, between these two conditions of disease cannot possibly be arrived at, in most cases, from the examination of the urine solely; but against this may be set the weighty support which the diagnosis derives from the collateral circumstances. Amyloid degeneration occurs almost exclusively among cachectic individuals, especially those who have been reduced by protracted purulent discharges (as from caries of the joints or of other spongy bones, pulmonary consumption, extensive skin ulcerations, or chronic ulcers of the intestines), or else by syphilis. Amyloid degeneration is the consequence of the cachexia, and occurs, therefore, in persons with poor, generally watery blood. Hence it is that in many cases we find it associated from the first with dropsy, or else entailing this condition at no distant interval of time. In amyloid degeneration a large portion of the arteries throughout the kidneys, with the Malpighian tufts attached to them, likewise lose their permeability. But, for all this, it seldom happens that increased pressure occurs in the arterial system, because, directly the blood-pressure begins to increase, the excess of water in the blood is pressed through the walls of the vessels, and produces dropsy. For this reason, too, we have neither dilatation nor hypertrophy of the heart, hypertrophy failing to develop itself because the blood itself lacks the very histological elements which are required for its production.

Still, I again repeat: without repeated and accurate examinations of the urine it is not every case of kidney cirrhosis that will be detected. Nor must we forget that the urine furnished by these organs will not always contain albumen; this element, however, will only be absent temporarily, as for example in the urine secreted during the night, or for a longer period under conditions of cardiac debility. *Regular diurnal examinations of the urine extended over a considerable time, and close attention to the state of the heart, form the groundwork upon which the diagnosis must be based.*

Treatment.

Considering the circumstances under which the greater portion of all the cases of genuine contracting kidney come under medical notice, it is evident that the prospect for therapeutic results must be a cheerless one. In point of fact such medical experience as we at present possess has furnished us with no means nor methods calculated to alter this discouraging view of the matter. If commencing dropsy, or the first symptoms of uræmia, or the impairment of vision provoked by the retinal affection, first indicate the presence of this insidious renal disease, then, certainly, we can no longer expect to preserve the patient from an early grave. Even were we possessed of the means to arrest the process of destruction, the help to be derived from them would then be all too late, since too much renal tissue would have been already destroyed for the compensatory measures still in operation to provide permanently against the actual defect of the renal secreting tissue. These very compensative measures, too, are in themselves departures from the normal condition of affairs which are replete with danger. The increased arterial blood-pressure threatens death by apoplexy, and this the more the longer it has endured; and who can entertain a doubt but that the constant loss of albumen, associated with the excess of pressure through the aortic system, must finally lead to the impoverishment and degeneration of the quality of the blood, and in this way impair the general nutrition.

How will it be, however, if the renal affection can be recognized at its first commencement? Here again the question that confronts us is, whether the destructive process of contraction must of necessity advance, whether under any, and if any, under what conditions, it can be arrested. Our present experience does not enable us to respond. We can only hope for an answer to this question when, by means of more carefully instituted clinical investigations upon the renal functions, we shall have presented to our observation more numerous instances of this disease at its commencement, and when we shall have succeeded in following the further progress of a larger number of such cases; for this question can only be decided empirically. It is true that a great practical difficulty stands in our way—the fact, namely, that the patient himself feels so well (and this often in spite of the progress of this renal malady) as to be little inclined to submit himself to a long-continued and disagreeable series of observations on the part of a doctor. I have rarely succeeded myself in keeping patients of this kind in my Clinic for more than a few months, whereas the object we strive after could be attained only by a year's observation.

Furthermore, the analogy between the course of this disease and that of similar processes of disease in other organs affords us no data upon which to determine the possibility of arrest in the contracting process once begun in the kidneys. The liver is the only organ in which an exactly analogous proceeding is observed. But we scarcely ever recognize liver cirrhosis until its terminal stage; indeed, the means that we possess for making an early diagnosis of this latter affection are almost poorer than those at our command for the early recognition of the analogous renal disease. Hence, we know no more of the curability of cirrhosis of the liver than we do of that of the kidney.

Lastly, we possess no exact knowledge of the actual causes of this kidney degeneration. Therefore, in individual instances we cannot even say whether they are removable, or whether from their nature they must of necessity continue in operation until the work of destruction is accomplished.

Thus, even if the disease is recognized at its very beginning, we therapeutists stand not only without any empirical guides, but without any reliable data to justify rational deductions. Indeed, the measures that I have undertaken for the purpose of arresting the progress of contraction, when recognizable, cannot be regarded as anything more than a cautious groping in the darkness which envelops the subject.

In laying down a plan of treatment for the cases of renal contraction that were recognized by me in a comparatively early stage, I have taken into consideration especially the dangers threatening my patients upon the one side from excessive arterial blood-pressure, and upon the other from anæmia and every other source of debility. Such patients who submitted themselves to my care at the hospital *I endeavored to guard from everything which might excite the action of their hearts in any unnecessary way.* I allowed them, indeed, daily exercise in the open air, but rapid or long-continued walking, as well as every sort of bodily exertion, was forbidden. The *first and foremost hygienic prescription for these cases*, so far as possible in private practice too, should be *rest in bed and the avoidance of all exertion and excitement in the pursuit of their business.* So long as they continue pretty strong I forbid my patients coffee, tea, and every alcoholic drink, on account of their influence upon the heart. On the other hand, I consider it necessary from the first to maintain the general nutrition and strength by an abundant meat, milk, and mixed vegetable dietary, because any failure in the nutrition at large holds out the prospect of degeneration in the hypertrophied heart, and renders the premature insufficiency of its compensatory action possible. As a matter of course, in this renal disease, too, proper attention must be paid to the skin. I enjoin warm clothing, direct flannel underclothing to be worn, and order frequent warm baths.

Starting on the supposition that, whatever preconceived opinion we may entertain of the nature of the process of renal contraction, we must admit that in every instance we have to deal with a growth of interstitial connective tissue which exercises a prejudicial effect upon the true glandular cells, I aimed to restrain this process of proliferation, and with this intent turned my attention to the employment of iodide of potassium. This substance recommends itself to us in so many cases of hyperplastic connective tissue growth that it appeared to me to deserve more confidence than any other medicament in this particular affection likewise. *I give iodide of potassium in solution to the extent of* 1.5 *to* 2 *grms.* (*from twenty to thirty grains*) *daily, and continue the use of this salt for an indefinite period*, and I can assure my readers that I have never seen any prejudicial effects from the use of this substance taken uninterruptedly for many months.

As to any direct influence of the drug upon the quantity or quality of the urine (except showing its own presence), I have remarked none whatever. It would perhaps be better to recommend patients to take the iodide of potassium while fasting early in the morning, since it is absorbed more readily by an empty stomach, and would thus be conveyed in more concentrated solution to the kidneys.

Unfortunately, the patients whom I treated upon this principle before the occurrence of any threatening symptoms, before their exhibiting any trace of dropsy or of uræmia, withdrew themselves from my observation too early, so that I cannot yet venture to form an estimate of the ultimate results of this method of treatment. In one of them, after being treated for four months, the albumen had as good as disappeared from the urine, for it was only in occasional specimens of his water that a slight opalescence could be obtained by boiling. The amount of urine passed by him during twenty-four hours fell from over 2,000 c.c., on his entrance to the hospital, to 1,400 c.c., or 1,500 c.c. on an average.

I never expected any particularly good results from any other line of treatment addressed directly to the renal malady, but I have tried several substances which are excreted from the system by the kidneys, and from whose employment one might therefore be tempted to anticipate some effect upon the renal functions. Thus I have, in particular, given various drugs containing tannin for some time without perceiving any consequences therefrom.

In the further destructive progress of the complaint it certainly happens that, aside from the *indicatio morbi*, many symptoms indicating particular remedial measures arise, and, in following them out, although we may not arrest the course of the disease in the kidneys, we may in some cases postpone its destroying effects for a season.

Signs of extreme elevation in the aortic blood-pressure, such as *palpitation*, *oppression of breathing*, *giddiness*, *roaring sounds in the ears*, demand complete rest and strict fasting on the part of the patient; an ice-bladder may be put over the heart, and its too energetic action may be quieted with digitalis or by narcotics (acetate of morphia, one and a half grains, dissolved in one and a half fluidounces of cherry-laurel water: a teaspoonful every three hours). Should distinct congestion of the head manifest itself in any such attack, I think a *moderate venesection* is indicated. As a rule, the patients who complain of these symptoms are still in such good condition as to bear a moderate loss of blood well. But patients in whom these symptoms have once occurred must be on their guard ever afterwards, for they return very easily, and always hold out the prospect of eventual apoplexy. Generally speaking, however, such occurrences as these are seldom the source of anxiety to the doctor, for the patients who are thus threatened usually submit to the inconvenience with an indifference that is scarcely conceivable. It is not until the later stages of the disease are reached, and after the symptoms suggestive of blood-pressure have long since made way for a totally different condition of things, that we learn sometimes, in our inquiry into the patient's history, that attacks of this nature have been previously experienced.

Anæmia and *commencing failure of strength* far oftener require medical attention and treatment, and this will have to be undertaken in different ways, according as the anæmia is the consequence merely of persistent losses of albumen in the urine, or is produced by some disorder of the digestive system—by dyspepsia or by troublesome vomiting. Individuals of the working classes frequently come into the hospital because, by reason of their strength failing them, they are no longer equal to the accomplishment of their ordinary day's labor ; they look pale and pulled down, but can give no reason for the wretched condition they are in, for, as they often positively assure us, their appetites are good, and they digest their food without any discomfort. Still, they have often noticed that for some little time there has been some slight œdema of the feet and ankles—an anasarca, however, that has subsided during the night. We examine them, and find that the left ventricle is hypertrophied, and that the urine contains albumen. Under the influence of rest and good nourishment in the hospital they often improve with remarkable rapidity, look well and fresh, the œdema of the legs subsides, not to reappear again ; but the urine remains albuminous.

I fancy that patients of this class, from the mere violent action of their hearts during the accomplishment of their laborious work, must lose more albumen in their urine while they are thus engaged than when they are quiet in the hospital, and that this very loss of albumen may be the sole cause of their anæmia and loss of power. The fact that these very same people, after being some time in the hospital and becoming stronger again, and then going about more than on their first introduction to hospital treatment, again excrete more albumen than they did at first, nowise contradicts my surmise.

The curative influence of rest and good nursing may be essentially assisted in such cases by the employment of iron.

Medical treatment is far less potent against the second source of anæmia referred to in this malady, *against the dyspepsia, and least of all against the vomiting;* for these symptoms must be regarded as the results of a blood rendered impure by excrementitious matters, and, therefore, as signs in themselves of an insufficiency in the renal secreting powers which can frequently never be repaired.

The ordinary remedies which one employs to improve the digestive powers fail us altogether. The appetite is not to be restored by bitters; and the employment of hydrochloric acid and pepsine at meal-times, from which I have often observed the promptest good results in the dyspepsia of anæmic subjects, usually leaves us in the lurch in this particular affection. Care must be used in the selection of the different articles of food, among which occasionally the most highly seasoned will be supported best, and good strong red French wine should be taken at meal-times, when the first signs of cardiac debility have established themselves. In other instances we may be compelled to put the patient upon an exclusively milk diet, because this is borne best.

The obstinate vomiting of the later stages of the complaint is *a most intractable torment.* It occurs very often before breakfast, while the stomach is still empty, and then, as I have frequently observed, it weakens the patient much less than when it takes place in the course of the day, and especially after meal-times. The usual treatment by effervescing mixtures, ice, etc., as a rule, does nothing to arrest it; but I have occasionally had successful results from tincture of iodine (two drops of the tincture in a tablespoonful of water), and from creasote too (one drop in a tablespoonful of water), directing these to be taken the instant the feeling of sickness comes on.

As soon as symptoms of general debility appear, constant attention must be paid to the *heart*, upon whose working powers the portion of the kidney that remains is absolutely dependent for means to carry on its functions sufficiently. So soon as signs of cardiac debility present themselves, the pulse losing its tension and the heart's sounds becoming feeble, the use of stimulants is indicated in addition to a dietary as strengthening as the powers of digestion in each particular case will support, and ferruginous preparations. Of all stimulants, good red wine has appeared to me the best for this purpose. I consider it advantageous too, under these circumstances, to give the iron in the form of an ethereal tincture.

Among other symptoms the next I shall refer to is the *dropsy*. In genuine contracting kidney this scarcely ever becomes the object of special treatment. It rarely reaches such importance as to be dangerous in itself, or even to be a very troublesome incident. Should dropsy occur at an early period of the disease, in consequence of temporary weakness in the heart's action, it will subside with the removal of this state of things, and sometimes does not return again for years. If, on the other hand, it presents itself towards the end of life, coincidently with the final marasmus, one never gets quit of it ; for patients who are brought thus low cannot support diaphoretic treatment up to the point where it becomes effective, and still less will they endure purging.

It would only be in the rarer cases of extreme anasarca, such as I have only met with once in renal atrophy (then combined with extreme fatty degeneration of the hypertrophied heart), that I should resort to mechanical evacuation of the fluid by punctures or scarification.

The treatment of *uræmic attacks*, which sometimes occur in the most violent form of epileptiform convulsions, even at a time when the general condition of the patient is still good, and when he appears to be in complete possession of his powers, the process of renal contraction being still far from advanced, requires the use of the most energetic measures, because of the immediate danger to life connected with them. Just in these very cases, however, which we are considering, difficulties of a peculiar kind present themselves in the selection of the measures we should employ to obviate the immediate peril, because our knowledge is so incomplete with regard to the actual cause of the fits. The old empirical practice, by which every seizure of this nature was treated by bloodletting and narcotics, has repeatedly failed me when applied in these attacks of acute, but, so to speak, premature uræmia, in persons with atrophied kidneys. When I ask myself how it comes to pass that *venesection is not attended by as good results in these cases as it is in the eclampsia of pregnancy*, I fancy that not unlikely the reason for such conflicting results lies in the dissimilar effects of blood abstraction upon dropsical and non-dropsical subjects. Pregnant women who are eclamptic are frequently dropsical. The premature uræmic attacks in contracting kidney happen to individuals, on the contrary, who are not dropsical. Now, the first effect of venesection, under all circumstances, will be a diminution in the general blood-pressure, and there must next follow an increased reabsorption of fluids from the tissues, and hence an alteration of the constitution of the blood, its serum, at all events, becoming more aqueous. Still we cannot tell what else besides water may not enter the blood in the increased reabsorption of the tissue fluids in particular instances. In dropsical persons there will be decidedly less solid material thus taken up again, because the dropsical fluid lying in the interstices of the subcutaneous cellular tissue, which is the first thing absorbed into the blood, contains very little solid matter. The composition of the fluids thus reabsorbed is altogether different when no dropsy is present. Then they will contain much more of the products of tissue metamorphosis, and will therefore carry those mischievous substances into the blood, to whose introduction, as I fancy, the dangerous state of things we are combating is attributable. Nevertheless, venesection, by effecting a general dilution of the blood, may reduce the total amount of this same mischevious material in it, and thus its employment even in these cases of uræmia may be desirable or allowable.

Theoretically, *the substitution of healthy for diseased blood*, by means of transfusion after antecedent bleeding, might be considered as greatly preferable to simple venesection. But the question of whether complete substitution is really feasible in this way appears to me as yet by no means quite decided. August Stoehr[1] performed transfusion in three cases of acute uræmia in chronic parenchymatous nephritis. It is true that none of them survived; still, in the first case, which succumbed to death by suffocation thirteen days after the operation, in consequence of copious effusion into all the serous cavities and of hepatization of the bases of both lungs, the immediate effect obtained in controlling the uræmic symptoms was very remarkable. Stoehr also relates a case in which blood substitution by transfusion was carried out by Belina-Swioutkowsky with the best and most permanent consequences, in a pregnant woman suffering with eclampsia and albuminuria.

It is self-evident that the sedatives nowadays so largely employed, and especially chloral hydrate, maintain their full value in the acute uræmia of the renal disease here under consideration.

In the intervals between the convulsive paroxysms in patients having severe uræmic fits, it becomes important to introduce as much water as possible into the system, in order still further to thin the blood by this means, and obtain a more complete washing out of the kidneys.

Against the chronic uræmia which sets in as the ultimate result of absolute deficiency in the amount of kidney substance, we possess no curative means whatever. The position is one entirely impregnable by physic. It is, of course, unnecessary to say that inflammatory effusions into the serous cavities may, under some circumstances, require emptying by operation.

5. Amyloid Degeneration of the Kidneys.

This peculiar form of degeneration also attacks the kidneys, and invariably invades both together, and extends diffusely through them. It is attended by albuminuria, and ordinarily in its later progress by dropsy, and for these reasons was regarded by the older writers merely as a peculiar form of Bright's disease which occurred in cachectic subjects; beyond this acquaintance with their genetic differences, nothing was known of the anatomical features which serve to distinguish this form from other forms of renal disease.

---

[1] Deutsches Archiv für klin. Medicin. Bd. 8. S. 467.

One of its anatomical peculiarities was first pointed out by Rokitansky in the first edition of his pathological anatomy in 1842, when he distinguished it from his other eight forms of Bright's disease as the lardaceous kidney. At a later period (in 1853) Meckel discovered the property possessed by this degenerated tissue, called lardaceous by Rokitansky, of *presenting a peculiar reddish brown coloration when it was touched with iodine, and of becoming violet upon the further addition of sulphuric acid.* He distinguished certain varieties or modifications of the disease by the degrees of coloration the tissue underwent, and thought that the degeneration was the result of a deposit of cholestearin in the affected tissues. Virchow[1] showed the incorrectness of this view, and considered the substance which showed this chemical action as being chemically analogous to vegetable cellulose, because it resembles the latter in this one particular, and hence he called it *amyloid matter*. Kekulé was the first to show that Virchow's views of its chemical nature were incorrect, and that it was rather a nitrogenous body, and in its elementary composition more fitly reckoned amongst the albuminates. Kuehne and Rudneff[2] especially brought forward the distinction between this and other albuminous substances, namely, that *amyloid matter was insoluble in gastric juice.*

Traube first taught how amyloid degeneration of the kidneys might be distinguished clinically from the other forms of renal disease associated with albuminuria.

Amyloid degeneration of the kidneys is nearly always the result of some previous or still existing process of disease, which more or less profoundly involves the nutrition of the body at large; and other organs besides the kidneys—the spleen, the liver, the abdominal lymphatic glands, the blood-vessels of the mucous membrane of the intestines, as well as those of various other organs—are probably without exception the seats of similar degeneration. *Amyloid degeneration of the kidney is therefore invariably the local manifestation of a general constitutional disease.*

---

[1] Virchow's Archiv. Bd. 6. u. Bd. 8.

[2] Ibidem. Vol. 33.

Etiology.

First among the causes which bring about the disordered state of nutrition (cachexia) that prompts amyloid degeneration of the kidneys, as well as of other organs, must be mentioned all those affections of various parts which are associated with prolonged suppuration. It at once becomes a question whether the purulent process merely gives rise to this particular cachexia by the persistent drain or waste of nutrient juices involved. I employ the term cachexia designedly, for it is not merely a question of marasmus. I never found kidneys in a state of amyloid degeneration in the bodies of diabetic patients who had died in conditions of most extreme marasmus, nor in the emaciated corpses of persons who had succumbed to stricture of the œsophagus. Yet these slowly fatal processes of disease had afforded time enough for developing the degeneration in question.

Again, patients who have amyloid disease are by no means invariably the victims of marasmus. Cohnheim found the kidneys of a soldier, who had been wounded in the field, and who died of his wounds two and a half months later, in a state of amyloid degeneration. I myself saw a woman who died from the effects of post-partum pelvic abscess—the abscess making its passage upon one side into the bladder, and perforating through into the rectum on the other—in whom the symptoms of this destructive renal malady were instituted at a time when her general condition as regards nutrition was very good, and her strength, too, was unimpaired. Not a few of my patients have been able to follow their occupations, and these in part laborious ones, at a time when this renal affection was discerned by me by symptoms of a most unequivocal kind.

My present experience compels me to conclude that, although every sort of bodily constitution admits of the possibility of amyloid degeneration of the kidneys and of other organs, as soon as the individual in question is the victim of an affection of any organ connected with chronic pus formation, which may serve as the special occasion for this degeneration, still particular anomalies of constitution (or diathesis) especially favor its development. As such constitutional predisposing anomalies I may specify *scrofula*, *chronic tuberculosis*, *inveterate* and *hereditary syphilis.*

Furthermore, according to my own experience, it is by no means a matter of indifference in estimating the probability of the appearance of amyloid sequelæ in the large abdominal glandular organs, what the original tissue or organ was in which the chronic pus-forming process of disease had its seat. *Amyloid disease with decidedly greatest frequency complicates those suppurative processes which are associated with actual ulceration*, *and*, *therefore*, *with molecular necrosis of the tissues.* They are hence associated with caries of the spongy parts of bone, and with extensive ulcerations of the skin and of the mucous membranes. Perhaps *the predisposition of scrofulous*, *tuberculous*, *and syphilitic individuals to amyloid disease* depends chiefly on the circumstance that the above-named affections are so regularly associated with chronic ulcerations of the bones, the skin, and the mucous membranes.

In the development of amyloid sequelæ it does not seem to be a matter of indifference, either, in what region of the body a process of chronic ulceration has been set up. At least I believe I may assert that extensive ulcers of the skin entail amyloid disease of the large visceral glands with especial frequency when their situation is upon the leg, be the cause of the ulcer itself what it may.

And I can positively affirm that *ulcerations of the intestines provoke amyloid disease more frequently than ulcers situated on any other mucous membrane*, and this, indeed, independently of the original etiology of the ulcer, which requires only to be of a chronic nature. It is certainly worthy of consideration, in this connection, that ulcerations of the skin of the legs and of the mucous membrane of the intestines exceed the ulceration met with on other cutaneous and mucous surfaces, both in the extent of surface they offer and the length of time they last.

I must further call attention to a fact which appears to me worth noticing, and is perhaps calculated to pave the way towards more clearly understanding the relation, obscure as it is, which obtains between chronic processes of ulceration and the amyloid degeneration of the organs above mentioned. In the great majority of instances of amyloid disease which I have seen, the original focus of infection was so situated as either to be directly open to the external air, whether by continuous extension of surface, or by fistulous passages, or else to communicate with cavities containing gas, which opened indirectly to the atmosphere, as, for instance, the intestines. Caries of the vertebræ may lead to extensive destruction of tissue resulting in the formation of enormous burrowing abscesses, and yet amyloid degeneration of the viscera does not ensue so long as the pus remains pent up in a large abscess cavity, and has no access to the air; in fact, these abscesses may last for several years, as I have recently had occasion to observe in two instances in which the enormous collections of matter were ultimately completely reabsorbed, without entailing amyloid degeneration. Should the abscess, however, open externally, and the contents of the cavity be exposed to the atmosphere, one has not, as a rule, long to wait before albumen appears in the urine, the sign of commencing amyloid degeneration of the kidneys. Similarly an *empyema* may endure for years in a pleural cavity without the abdominal glands becoming amyloid; but directly thoracic fistulæ are formed, either artificially or spontaneously, we must take good care that the further secretion that takes place is never allowed to stagnate, inasmuch as the pus within the pleural cavity is now exposed to the external air, and, if it be allowed to stagnate, we may reckon with the greatest likelihood upon the speedy supervention of amyloid disease. *Chronic tuberculosis* may effect large deposits in the lungs and lead to most extreme marasmus, without our being able to discover a trace of amyloid disease in any organ whatever of the body. *But if the tuberculosis causes large cavities by a process of ulceration*, the degeneration we are speaking of seldom remains absent, if the disease be not too rapid in its course to admit of its development. I have frequently observed it to ensue likewise as the consequence of very extensive bronchiectasis. I must not, however, conceal the fact that I have seen amyloid disease of the kidney commencing at adult age in individuals who had suffered from chronic (scrofulous) affections of their joints and bones in early life, but in whom no free discharge of pus had ever been established. In two such cases the subjects had enjoyed apparently good health for several years.

The sum of the facts above collected compels us to assume that it is not the failure of the general nutrition, in and of itself, nor the formation of pus, with the consequent waste of nutritive juices, that suffices to provoke amyloid degeneration of the kidneys or of the other abdominal organs; but that *there must exist some special cause, perhaps some chemical agent*, which is formed quite preferentially in these particular foci of disease where molecular destruction of the elements of the tissues is brought about by necrobiosis. It may be that some substances obtained from the atmosphere, whether it be oxygen or some bodies capable of generating fermentation, may contribute toward the development of this cause. We may imagine that this particular noxious agent is conveyed from the seat of its formation into the blood, and thence deposited in the tissues that become the seat of amyloid degeneration, or else that the mere contact of the substance provokes those remarkable metamorphoses in the walls of the blood-vessels by which amyloid degeneration always begins.

In point of fact, there has been no lack of endeavors to discover this amyloid substance in the blood, and indeed a physician of Toronto in Canada professes to have found it.[1] But no other investigator has come upon it as yet.

The doctrine which led to the performance of these experiments, namely, that *the amyloid substance was contained already formed within the blood*, and was deposited thence within the walls of the vessels, is opposed by the extremely weighty circumstance that amyloid degeneration is far from being found in every blood-vessel of the human body, but invariably occurs only within certain more or less narrow limits. Now, if it were true that the amyloid substance was deposited from the blood, it would be incomprehensible why the contractile cells only of *the arteries of the digestive tract*, *of the spleen*, *the liver*, *the kidneys*, *the supra-renal capsules*, *the lymphatic glands*, *and the thyroid gland*, should be the uniform seat of this deposit, while all the other vessels of the body should be free from it. Still it is equally difficult, in the present state of our knowledge, to explain by any other hypothesis the fact of the vessels within this limited extent experiencing so peculiar a metamorphosis.

---

[1] *Rud. Virchow*, Die Cellularpathologie. 4th Auflage. 1871. S. 443.

Lastly, I must remark that amyloid degeneration now and anon occurs in the large intra-abdominal glands of persons in whom no conditions whatever can be discovered which might have caused the cachexia that underlies it, in individuals who have never in their lives suffered from chronic suppurations, or syphilis, or bone disease, or malarial fevers, and in whom, therefore, the amyloid affection has occurred, so to speak, as an entirely spontaneous idiopathic disorder of nutrition.

If it ever be allowable to make a diagnosis of amyloid disease of the abdominal glands merely upon the clinical symptoms, the following case may be considered as being such an one, as well as serving by its example to show that amyloid disease may under certain circumstances recede, or at least need not necessarily proceed to a fatal issue.

Case XXVIII.—J. H. G., a weaver, forty-four years of age, came to the consulting-room for out-patients in our Clinic, from a neighboring village, on the 8th of July, 1863. His face had a particularly pale, dirty, sallow hue, and he was complaining of œdema of both lower extremities, which, according to him, had existed for a full month past. His health had previously always been good; especially had he never suffered from intermittent fever or syphilis.

G.'s general nutrition upon this his first appearance was still tolerably good, as also was his strength; he had made his way on foot from the station to the hospital in spite of his swollen legs, a good quarter of an hour's walk for a vigorous walker. His abdomen was considerably distended, in part from an appreciable quantity of free fluid present in it, but partly and principally, too, from very considerable enlargement of both liver and spleen. His liver reached down far below the right margin of the ribs, and its left lobe completely filled the epigastric region. In the median line the liver dullness measured 16.5 cms., in the mammillary line 12 cms., and in the axillary line 7 cms.; the left lobe extended 9 cms. across the median line. The liver felt hard; but its surface was perfectly smooth. On the left side the spleen, which was very hard too, came below the costal arch; taken diagonally from above downwards, the longest diameter of the splenic dullness measured 17 cms., the shorter transverse diameter 14 cms. The patient's pale yellow urine contained a good deal of albumen. In the sediment of the urine we found some scattered small hyaline casts, to which were attached some highly refracting cells of the size of renal epithelium. Some other cells like these, but somewhat larger, were also noticed either lying singly or arranged in rows, and distinctly nucleated. The remarkable lustre of these unusually large cells caused me to add a watery solution of iodine to the microscopic preparation, when both the cells that adhered to the casts, as well as the others, assumed a bright reddish brown color, while the substance of the hyaline casts only assumed a faint yellow color. The further addition of sulphuric acid changed this reddish brown to a somewhat dirty violet hue. The patient still avowed that his appetite was good and his digestion was unaltered, although the grayish white color of his fæces had attracted his attention. Still there was no icterus. The heart and lungs appeared sound. As the result of my examination of him, I thought that although no etiological antecedents of amyloid degeneration of the intra-abdominal viscera could be discovered, still the changes that had been recognized must be referable to such degeneration. He was ordered iodide of potassium and tincture of iron.

This treatment was so far successful that, in the course of the summer, considerable diminution in size took place in both liver and spleen, the albumen contained in the urine diminished, and the patient's aspect and strength improved to such an extent that he finally ceased his visits to the Clinic.

In December, 1864, he appeared again to bring a sick daughter to me. He himself continued to feel well, although the splenic dullness in its long diameter still marked 13, and in its short diameter 11 cms. The spleen and liver, too, still extended on each side considerably below the costal arches, and both organs felt remarkably hard and firm. The urine still contained albumen, although I could find neither casts nor any of the large lustrous nucleated cells in it which had never failed me in any portion of his urine that I had examined during the previous year.

In the years 1865, 1868, and 1870 respectively, I saw the man once each year and examined him, satisfying myself of the slowly progressing diminution that was taking place in both liver and spleen. But even on the 19th of April, 1870, his urine still contained some albumen. The patient then passed out of my sight for four years until, by chance, I met him one day, when at my request he came up for another examination at my Clinic.

On the 25th of April, 1874, the following note was taken: general nutrition good, rosy complexion, muscles powerful, heart and lungs sound, liver still reaching a little below the ribs and feeling somewhat hard. Liver dullness in median line 7; mammillary line 9; axillary line 5 cms. Splenic dullness from above downward 7.5, from behind forward 8 cms. The urine passed at the Clinic had a specific gravity of 1015; it presented an acid reaction, and did not contain a trace of albumen.

I should hesitate to adduce this case as a specimen of amyloid disease, and still more as an example of an at least comparative cure of this degeneration, if the characters of the cells in the urine had not, as I think, justified my diagnosis, and if I had not become convinced of at least the quasi-curability of this degeneration by later experience, in cases where the etiology as well as the symptoms have indicated the amyloid nature of the renal malady beyond any doubt.

Lastly, I must here call attention to one peculiar circumstance, viz., the exceedingly frequent coincidence of amyloid disease of the kidney with other renal affections. It follows, as a matter of course, from what has been said above, that pyelonephritis upon one side (ulceration of the pelvis of the kidney and of the kidney itself) may be accompanied by amyloid degeneration of the organ upon the opposite side, and that the latter would then be considered as the result of the former. But it happens remarkably often that *chronic parenchymatous nephritis and amyloid degeneration occur coincidently in both kidneys*, and in these instances both processes are apparently co-effects of one and the same cause, for our experience teaches us that chronic suppuration will frequently give rise to chronic nephritis alone without amyloid degeneration.

After what has been already said upon the etiology of this affection, it will be seen that amyloid degeneration of the kidney may happen at any period of life. Dickinson (quoted by Lecorché) says that he has never witnessed this affection during the early years of life, up to the age of five; again, it does not appear to occur in extreme old age. It has been noticed somewhat less frequently in women than in men. As the provoking causes of amyloid degeneration of the kidneys are common, so the disease, too, is of quite frequent occurrence.

Review of the Course of the Disease.

In the large proportion of cases, amyloid degeneration of the kidney occurs in individuals who, both previously and at the time when the renal malady commenced, exhibited symptoms of other grave disease; it further coincides regularly with amyloid disease of other vital organs; hence, it is scarcely possible to form an idea of the effect produced by the renal disease itself, or in any given instance to decide what part of the complex of symptoms presented we are to attribute to the fundamental disease, and what to the renal changes, and how much belongs, finally, to the analogous degenerations taking place in the liver, the spleen, the lymphatic glands, etc.

With but few exceptions, this renal disease attacks individuals who are weakened by one cause or another, and very commonly there exists a greater or less degree of anæmia, even before the renal malady begins. Again, at the commencement of the degenerative changes, every symptom may be absent which could direct the attention either of the patient or of his doctor to the kidneys. *The morbid change develops itself quite insidiously, or, so to speak, without symptoms.* Any one who has not made it his rule, under all circumstances that appear at all suspicious, to examine the urine of his patients minutely and repeatedly, will either not recognize the disease at all, or will not perceive it until, some fine day, the patient surprises him by general dropsical symptoms, which, for that matter, are by no means constant accompaniments of renal amyloid degeneration, and perhaps in some cases are dependent rather upon the fundamental cachexia than upon the kidney malady.

The only immediate *results of the renal disease* which we can claim with certainty as such are the *functional disturbances* which are connected with it. Therefore, we will consider them first.

In not one, perhaps, of the various forms of diffuse renal disease does the *urinary secretion* exhibit in different cases such great differences in relation to its quantity and constituent elements, and in no other kidney affection does the amount and chemical composition of the renal secretion demonstrate such great and repeated variations in one and the same case as we observe in amyloid degeneration of the kidneys. Still, the rule may be held to obtain that *kidneys implicated in amyloid degeneration secrete abundantly.*

poly

Thus, for example, a woman who, after a post-partum pelvic abscess, acquired amyloid degeneration of the spleen and kidneys, passed, upon an average of twenty-three estimations conducted over a period of two months, a daily average of 1,580 c.c. of urine; and a month later, upon an average, it is true, of only five analyses, 2,100 c.c. A man, afflicted with the same renal degeneration, passed, upon an average of forty-seven estimations made in the course of two months, a daily mean average of 1,678 c.c. of urine. The *diurnal amounts*, however, fluctuated during this period between 450 and 3,400 c.c. The most extensive series of urine analyses was instituted upon the water of a Swedish laborer, who had suffered at nine years of age with ostitis of his right tibia, in consequence of which this bone became six centimetres longer than that of the opposite leg. At the date of his reception he was twenty-eight years of age. There existed recent periostitis of the right tibia and œdema of both legs, as well as of the scrotum. Upon an average of 135 estimations conducted over a period of five months, he passed a daily mean amount of 1,902 c.c. of urine (minimum 1,000, maximum 2,600 c.c.). Three months after his discharge he died. The post-mortem examination showed extreme amyloid degeneration of both kidneys. A fourth patient, in whom the renal malady succeeded spondylitis in both the cervical and the lumbar portions of the vertebral column, and who had a burrowing abscess in the neck, opening externally, may be taken as an example of a reversed condition of things. He passed only 481 c.c. of urine daily, upon a mean average of twenty examinations made during one month.

In many cases it is easy to demonstrate the existence of certain definite influences which account for this diminution in the urinary secretion, which is contrary to the rule, as well as for the remarkable daily fluctuation in the amount of urine secreted. Thus, patients who, before the renal degeneration began, suffered with profuse diarrhœa, invariably excrete small quantities of urine, and others in whom diarrhœa occurs during the progress of their renal malady, at once cease secreting much urine. In other instances, however, we are unable to discover any such reason for the temporary or persistent reduction of the renal secretion.

Such *extreme augmentation of the kidney secretion* as occasionally occurs in genuine contracting kidney, *I have never seen in amyloid degeneration*, and the amount of urine passed always diminishes towards the close of life, often falling to a few hundred cubic centimetres as the failure in the patient's strength becomes more marked, even complete suppression occasionally taking place some days before death. I have repeatedly known the urinary secretion, which had previously always been abundant, suddenly to become arrested altogether.

Thus, for example, the laborer S. during two months daily and uniformly passed 1,560 c.c. of urine, and upon the 8th of November, 1874, 2,470 c.c. of urine. During the last eight days of his life, from the 10th to the 17th of November, only 594 c.c. were secreted *in toto*, or a daily average of not more than 74 c.c.

The urine furnished by amyloid kidneys is always *clear*, *remarkably pale*, sometimes *almost as clear as water*, and only darker and occasionally clouded with urates when secreted in very small amount.

If *much* urine is passed, its *specific gravity sinks extremely low*. I have often examined such urine which hardly marked 1003. When, on the contrary, *very little* urine is passed, its specific gravity may *greatly exceed the physiological standard*, going up to 1030 and over.

Hence, the percentage of solid contents, and especially of urea, contained in the urine, will vary much in different cases, and in the same case at different times. In all instances where there is polyuria the percentage of urea contained in the water will be extremely small, falling often below one per cent. When, on the other hand, small quantities of urine are secreted from the very commencement, the percentage of urea may be abnormally high, rising to four per cent. This is, however, not the case when the daily quantity of urine, by reason of general failure of strength, has been reduced to a very small amount. Thus, in some cases, we found that during the last days of life, when the quantity of urine for the twenty-four hours scarcely reached 100 c.c., the amount of urea contained in it was not more than from one to two per cent.

The total amount of urea excreted by kidneys that are the subjects of amyloid degeneration *depends much less upon the actual state of the kidneys themselves than upon the activity of the general tissue metamorphosis throughout the body*, which again is itself more affected by the state of disease of other organs than by that of the kidneys. Thus, it may happen that highly amyloid kidneys quite a short time before death excrete considerable total amounts of urea, and that the diurnal quantity of urea excreted at an earlier period of the affection may, under some circumstances, exceed ordinary physiological amounts. Over forty grammes of urea is a quantity that I have often found furnished during twenty-four hours by amyloid kidneys, in cases, too, in which the correctness of my diagnosis was confirmed by subsequent post-mortem examinations.

The previously mentioned Swedish laborer, for example, during the five months he was under our observation, excreted, as the average of thirty-one examinations, 32 grms. of urea daily (minimum 19.5, maximum 44.0 grms.).

The *chlorides* and *phosphates* contained in the urine are liable to fluctuate in their amounts just like the urea. The total sum of the chlorides excreted may still in particular cases resemble the normal averages.

In proof whereof I may again refer to my Swede, who, as the mean result of thirty-one analyses extended over five months, excreted 13.0 grms. of chlorides daily in his urine (the minimum being 10.25 and the maximum 16.0 grms.).

The amount of *phosphoric acid*, nevertheless, in the few instances in which we estimated it, was invariably found to be below the normal standard.

In every specimen of urine from an amyloid kidney that I have examined I have found albumen; but this again differs very much in amount in different cases, varying from the slightest cloudiness upon boiling up to three per cent. and over.

I regard the presence of albumen in the urine as quite an indispensable element in the diagnosis of amyloid degeneration of the kidneys, without which the recognition of the affection during life is, in my opinion, impossible.

Still, it may be an open question whether albumen passes into the urine at the very commencement when the amyloid degeneration of the walls of the secreting vessels is just beginning; but this I am sure of, that I have never yet found distinctly marked amyloid disease of the kidney in the bodies of persons whose urine during their lifetime had been tested by me for albumen without its being discovered. Considering the extreme care with which for many years past the examination of urine in our Clinic has been prosecuted, and the autopsies performed, it is impossible that the fact should have escaped us of the non-appearance of albumen in amyloid degeneration, if this had been the case. And, upon the other hand, it has been established that in the cases that terminate fatally the albuminuria persists up to the close of life. Even in the smallest amounts of urine which are passed during the last few days before death by patients exhausted in the most extreme degree, albumen is still to be found. I lay great stress upon this statement because M. Lecorché,[1] in opposition to the quite universally received opinion, asserts that amyloid degeneration of the kidneys in and of itself never does cause albuminuria, but that the appearance of this symptom in amyloid disease should be accepted as sure evidence of the affection being complicated either by parenchymatous or by interstitial nephritis. The statement is one which I must contradict in a most positive manner. I am too well aware of the extreme frequency of these complications of amyloid degeneration from my own multiple experience of them to overlook their occurrence in the dead body; and I can for this very reason say positively that in cases of pure amyloid renal disease albumen is a constant ingredient of the urine as soon as the degeneration has effected distinctly recognizable changes in the kidneys. Lecorché, it is true, also states that amyloid degeneration of the kidneys during its course is nearly always complicated with parenchymatous nephritis, and this assertion I must, according to my own experience, also deny. This opinion of Lecorché's may perhaps be explained by the circumstance that he has confounded the fatty destruction of the epithelium in amyloid kidneys with inflammatory infiltration and degeneration. At all events, his description of the microscopical appearances after parenchymatous nephritis confirms this surmise.

In the cases of amyloid degeneration of the kidney which I have, so to speak, seen in process of development—in patients, that is, whose condition permitted me to expect the advent of this renal affection—I have noticed that the albuminuria ordinarily made *very slow* progress; that the urine, passed as it usually is at first in very small quantity, contained only very scanty traces of albumen. This slight albuminuria is ordinarily not permanent at first. It lasts a few days, then disappears, again to recommence afresh, and finally to become permanent. *In most cases the percentage of albumen contained in the urine is at*

---

[1] L. c. p. 669

first considerable (from one to two per cent.), until, with the occurrence of the polyuria, which arises later in the disease, the proportion of albumen in the large quantities of urine passed may often not exceed a few parts per thousand. Thus it may continue to the end, the percentage of albumen not rising again, even when the propulsive power of the heart fails toward death, and the actual secretion of urine sinks to a minimum. In other instances I have observed that the quantity of albumen, which had been noticed to be small for some time, quite suddenly rose to a considerable percentage, and that this condition of things then remained permanent; but the total amount of urine passed then always remained abnormally small.

The *total loss of albumen* is just as dissimilar in different cases as is its percentage. In no small number of cases in our Clinic careful analyses of the urine were conducted, extending over a protracted period, and the results arranged in tabular form for each particular case. From a study of these tables, it appears that the *average daily loss of albumen* fluctuated between five and twenty-two grammes.

Our Swedish laborer lost during the five months he was with us, as the result of thirty-one analyses instituted during this period, a daily average of 19.49 grammes of albumen with his urine (minimum 12.95, maximum 31.92 grammes).

In the chapter in which I discussed the subject of albuminuria, under the General Symptomatology of Renal Diseases, I referred to Senator's statement that the urine in amyloid disease of the kidneys, besides containing ordinary serum-albumen, also contains globulin in comparatively large amounts, and that this circumstance might perhaps possess some diagnostic importance. Our own observations seem to confirm Senator's statement, and this is of all the more consequence because the greater portion of these examinations were made some years ago by Prof. Edlefsen in a series of cases of disease in which albuminuria had been noticed. Looking through our urine tables I now find that Edlefsen noticed specially large amounts of globulin in the urine of patients with amyloid disease. Our latest examinations of the urine furnish the same results.

I have only seen blood in the urine once in a case of amyloid kidney, but it was then present in considerable quantity. The case was that of a man who was broken down by syphilis, but after death we found thrombosis of both renal veins, besides the degeneration of the kidneys, the spleen, and the liver.

I have already remarked that the urine of patients suffering from amyloid disease of the kidneys is, as a rule, perfectly transparent, and deposits no sediment. It is therefore *exceedingly seldom that we find casts in it in any considerable number.* The circumstances under which we may find a considerable number of casts, and sometimes a very great abundance of them, are those in which the urine is secreted in scanty quantity, is of high specific gravity, and contains a great amount of albumen. But even urine of this kind more generally contains but few casts.

When large quantities of urine are passed, we often seek in vain for any structures of this kind, and the rare samples we discover after a prolonged search *are nearly all of the narrow and perfectly hyaline variety.* In those cases in which *the urine contains casts in large quantities, the number of wide casts exceeds the narrow ones, and* we also find *waxy refracting casts* and *yellow casts* in *largest number*, besides *some dark granular ones.* We have already noticed above that under such circumstances these wide casts occasionally assume a reddish brown color with iodine.

Thus much with regard to the symptoms which arise from the functional disturbances induced by renal degeneration. With regard to all the other symptoms of the disease, it is impossible to say in how much or in how far the renal malady is concerned in their production, or whether in any individual case they are exclusively or partly the result of the fundamental disease or of the simultaneous amyloid degeneration of other organs. Most of the persons who are affected with amyloid degeneration of the kidneys become dropsical; others remain perfectly free from dropsy to the end. Still, this is certainly not often the case. Grainger Stewart found that general dropsy occurred only six times in one hundred cases. The dropsy is ordinarily confined to the lower extremities and the abdominal cavity. The pleura and the pericardial sac, as Lecorché states upon Roberts' authority, nearly always remain exempt. Speaking generally, we are enabled to confirm the correctness of this by our own observations; still, exceptions to this rule do occur.

After it has once appeared, the dropsy often remains remarkably persistent, especially the *ascites*, and this even in cases where the urinary secretion is abundant. Lastly, it may happen that traces of dropsy show themselves before the commencement of the albuminuria, that is, presumably before the disease of the kidney has begun.

The peculiarity in the distribution of the dropsical effusions, differing as these do from those which complicate ordinary renal dropsy, their frequent demonstrable independence of the amount of water excreted by the kidneys, and their occurrence even before the kidney affection has commenced, all prove that *the dropsy of amyloid degeneration of the kidneys is not always the result of the retention of water in the blood in consequence of its defective excretion through the kidneys.* The renal disease in most cases certainly plays a subordinate part, although it does give occasion for the removal of still larger amounts of albuminoid substances from the blood, which is already poor enough in albuminoids, and hence does assist in promoting hydræmia.[1] The fact that in amyloid patients the dropsical effusions take place by preference in the abdominal cavity is thought by Roberts to be due to the disturbance of the circulation through the vena porta, which amyloid degeneration of the hepatic vessels and the swelling of the degenerated lymphatic glands about the portal vein entail.

In spite of the considerable loss of albumen associated in many instances with amyloid degeneration of the kidneys, *the renal affection really has small part in the production of the anæmia, the cachectic appearance, and the loss of strength* which we notice in the larger number of these patients. If the fundamental malady has not itself produced this state of cachexia, it is far more likely to be due to the simultaneous degeneration of the spleen, the liver, and, above all, the true digestive apparatus.

---

[1] Grainger Stewart examined the blood of an amyloid patient of his. The serum had a specific gravity of only 1018.5, and contained merely 6.16 per cent. of solids. But such single analyses of the blood as this prove nothing; its composition will depend upon circumstances, and will vary as much in amyloid cases as it does in every other case. I took some blood from a body directly after death, and found the specific gravity of the serum = 1028.23, with 13.65 per cent. of solids. The body contained amyloid disease of the large glands. Slight œdema of the lower extremities. Death by erysipelas. Woman aged twenty eight.

A very common and nearly always fatal symptom connected with the latter is *diarrhœa.* It usually resists all treatment most obstinately, and by the exhaustion which it causes greatly hastens death. Diarrhœa may result from ulceration, as in phthisis and syphilis, and this ulceration may have existed before the amyloid disease began, and indeed may have given rise to it. But a diarrhœa scarcely less troublesome and almost more destructive occurs with intestines which were previously intact, as the consequence of amyloid disease of the intestinal blood-vessels, and perhaps depends, too, upon stasis of the aqueous blood in the domain of the portal circulation, by reason of degeneration of the hepatic vessels. Then ulcers may also be formed subsequently through extensive necrosis of the mucous membrane.

Vomiting occurs in amyloid disease with far less frequency than diarrhœa, but then it is no less obstinate than the latter, nor less exhausting. The vomit consists, as a rule, of watery material, which ordinarily has a faintly acid reaction; still, in one case I found it alkaline, and smelling strongly of ammonia, as did also the fluid fæces passed at the same time. In both fluids my colleague Edlefsen showed by chemical tests that ammonia was present. In this, as well as in other similar cases, no gross lesion of the mucous membrane of the stomach could be seen after death. Was the vomiting, which in this case persisted up to death, of a uræmic character?

The other symptoms of uræmia are certainly most rare and exceptional in amyloid degeneration of the kidneys. Out of the large number of my own patients, one only died after a severe attack of epileptiform convulsions. Uræmic symptoms, too, were completely wanting in those cases which, even for days before death, did not pass a drop of urine (in one case for five days).

I have never known *death to occur by apoplexy* in amyloid disease of the kidneys, and *in one case only* of the many whose bodies I have seen examined was the *left ventricle of the heart found hypertrophied*—excepting, of course, the cases that were complicated with contracting kidney.

The absence of cardiac hypertrophy in amyloid disease might appear strange, since conditions are present that are exactly analogous to those which, at least, so far as I understand the matter, determine its development in contracting kidney, namely, obliteration of a large number of blood-vessels. But here the hydræmia which exists, as a rule renders the obstruction ineffective. As soon as any increased pressure of blood takes place in the vessels, the aqueous part of it passes without check into the interstitial connective tissue and the serous cavities. In this way any considerable increase of blood-pressure in the aortic system, and consequent dilatation and hypertrophy of the left ventricle, would be obviated.

The fatal issue which certainly attends the larger number of cases of amyloid disease is less frequently occasioned by complications of an inflammatory nature occurring in other organs than it is in any other renal affection. Purulent effusion into the peritoneal cavity is the only one of these complications that has presented itself to me with comparative frequency. Rosenstein makes the same observation. As a rule, the fatal termination is brought about by a process of gradual exhaustion, and this is certainly in most cases not due merely to the renal affection.

Here too, I must once more revert to the *possibility of recovery* from amyloid degeneration of the kidneys, and in so doing I shall entirely pass over those equivocal cases in which a temporary albuminuria arises in the course of chronic ulcerations and suppurations, an albuminuria that subsides with the removal of the provoking cause, and which one might be inclined to regard as some slight touch of the degeneration in question. The cases which I have in view are rather those in which the diagnosis was less dubious, and in which the renal affection, as urinary analyses showed, lasted for years. One such case I have already related above; I will give another one here.

Case XXIX.—Miss N. N., from Hamburg, came under my treatment as a private patient in 1868. The father of this young lady (she had lost her mother in childhood, of some complaint the nature of which was not known) was probably syphilitic, for he had several times been to Aachen to undergo the treatment there; he died shortly before her arrival at Kiel.

The young girl was considerably swollen with anasarca throughout the entire lower half of her body, and had ascites. Her face and hands were not œdematous. The dropsy had begun without any known cause three months before her arrival. Still the little woman had been poorly for several years, and, being supposed to have scrofula, had been sent to a variety of bathing-places. The lachrymal ducts were closed on both sides, so that her eyes were nearly always swimming with tears. There was a very remarkable abnormality about both legs: the legs, below the knee, were far too long in comparison with the thighs; both tibiæ were extremely bowed forwards, the anterior edge of each protruding as a sharp ridge beneath the tightly stretched skin. Measurements instituted at a later period showed the lineal distance between the external condyle of the tibia and the lower border of the malleolus externus upon the right leg to be 38 cms, and upon the left leg 38.5 cms., *while both femurs, from the upper border of the trochanter to the lower border of the external condyle, measured but* 35 *cms.* There was further a so-called false hypertrophy of the muscles of both calves, which, although imperceptible when I first saw her, made itself apparent afterward when the dropsy had subsided.

The urine was passed in moderate quantity, was dark colored, clear, and contained a great deal of albumen, although but few casts.

During the four months while she was under treatment, taking daily hot baths, and using iodide of potassium internally, her dropsy entirely disappeared. Directly the subsidence of the ascites rendered the examination of the abdominal organs feasible, it was ascertained that the spleen was considerably enlarged, so that its lower edge could be felt below the margin of the ribs on the left side.

During these four months the entire amount of urine passed upon twenty separate days, taken at distant intervals of time, was carefully collected and analyzed. The results of these analyses are shown by the following mean averages:

Amount per diem of urine	686 c.c.
" " " albumen	6.3 grms.
" " " urea	18.0 "

The largest amount of albumen was 1.56 *per cent., and of urea* 4.3 *per cent.* The specific gravity varied between 1018 and 1038. At the time she left, in October, the patient's own sensations were perfectly satisfactory: she looked rosy, but the urinary secretion remained entirely unaltered.

In July, 1869, the young lady returned again, and was admitted into my Clinic, where she remained for some months. She retained her healthy look, but since November of the previous year had become so deaf that she was compelled to make use of an ear-trumpet to hear with. The principal cause of her return, however, was her anxiety about some slight œdema of her legs. The spleen had diminished considerably since the previous year. The same treatment was adopted as before, and with similar, only still more rapid results.

In July, 1869, before the treatment was commenced, the patient passed 1,010 c.c. of urine daily, with a specific gravity of 1018 and containing 4.343 grms. of albumen, and 20.2 grms. of urea. At the time of her discharge in October we have the following mean result of six accurate analyses, the examinations having been instituted at considerable intervals of time.

Diurnal amount of urine	647 c.c.
" " albumen	3 grms.
" " urea	23.5 "

The highest percentage of albumen was 0.5 per cent., and of urea 4.5 per cent. The specific gravity fluctuated between 1018 and 1034.

When the patient returned to Hamburg in October, every trace of œdema had disappeared, and she was feeling perfectly well. From this date no further œdema showed itself.

In the year 1872 I saw this patient again; she had become almost absolutely deaf, and had suffered, as she told me, for some time with nasal catarrh, in consequence of which several portions of bone had been removed from her nose. The bony part of the septum narium showed a large deficit. At that time her urine still contained albumen. I am indebted to one of my Hamburg friends for making this examination.

In the summer of 1874 I learned from the same gentleman that Miss N. N. felt completely well, and that the latest urine he had examined no longer contained albumen.

I do not hesitate to reckon this case as an example of amyloid degeneration of the spleen and kidneys, and assume it to have been caused by the cachexia of hereditary syphilis. M. Lecorché, it is true, asserts that hereditary syphilis never induces amyloid disease because it never leads to ulcerations. I can refute both these statements by several cases which occurred in my own experience.

The hereditary syphilitic disease known in this country under the name of "Morbus Dithmarsicus" leads frequently to enormous destruction both of skin and bone by ulceration, and is by no means rarely followed by amyloid affections.

On the whole, then, I believe we may state that the issue of the forms of disease which are attended by amyloid degeneration of the kidneys depends much more upon the fundamental malady and the simultaneous amyloid affection of other organs than it does upon the renal disease itself.

The above statement holds good, especially as regards the *duration* of the kidney affection. Leaving entirely out of consideration the trifling commencements of this degeneration in which micro-chemical analysis alone can show us that any positive change in the kidney has taken place—a change which does not occur until towards the end of a process of disease which is in itself fatal—experience teaches us that, in well-established cases of amyloid kidney, *the duration of the disease, from the first appearance of the renal symptoms until death, may vary very materially.* Thus, in the body of a young girl of eighteen, who since she was nine years of age had been afflicted with syphilitic lupus of the skin of the face and neck, I found extreme amyloid degeneration of the kidneys four months after the first appearance of albumen in her urine. Against this may be set cases also of syphilitic origin which have lasted for several years. The two cases of recovery above related by me had lasted respectively seven and five years from the date when the renal malady was first discovered, to the time of the last examination, when the still perceptible amount of albumen contained in the urine made the continuance of the affection at this date at least probable. Grainger Stewart[1] communicates a case which lasted for nearly ten years. From my own observations I am satisfied that this renal degeneration progresses at very unequal rates in different cases, perhaps from dissimilar intensity in the provoking agents.

Review of the Post-mortem Appearances.

The circumstance that death often sweeps off patients who have amyloid disease of the kidneys by some other affection, just at the commencement of the renal degeneration, has facilitated the pathological investigation of this condition throughout the entire progress of its accomplishment, from its faintest beginnings to its most extreme development. The *slighter grades* of amyloid degeneration alter the aspect of the kidney and its other characters so little that this looks perfectly normal to the naked eye. It is not until the more advanced stages of the disease are reached that the whole organ appears altered in any remarkable or characteristic way. Still, "a positive diagnosis," says Virchow,[2] "can be made only by the application of iodine, and even then one must take care first to have washed away all excess of blood out of the vessels, for a vessel full of blood shows exactly the same coloration upon the addition of iodine as a vessel which has undergone amyloid degeneration would present."

---

[1] L. c. p. 152.

[2] Cellularpath. Vierte Auflage. S 446.

Kidneys which have undergone extreme amyloid degeneration are much larger and heavier than normal organs. The thinned capsule is easily stripped off, and after its removal the surface of the organ remains smooth and even, and has a remarkably polished appearance. The color of its surface is quite noticeably pale, at times almost white. Here and there occasional stellate veins are to be seen, filled with blood, their dark bluish red hue standing out in strong contrast against the pale background. The broadened cortical substance of the kidney exhibits the same extreme anæmia, and is equally pale colored, its pallor contrasting again in a lively manner against the dark reddish brown of the medullary cones. The peculiar polish upon the cut surface in the cortex is likewise remarkable.

A large, pale, anæmic kidney of this kind feels quite remarkably firm and tough. This fact, as well as the absence of the peculiar yellow coloration which one notices in the relaxed and swollen but equally anæmic kidney of chronic parenchymatous nephritis, distinguishes the pure form of amyloid kidney from the variety just described. In chronic parenchymatous inflammation, too, a higher grade of swelling is ordinarily induced.

If, now, we apply a watery solution of iodine and iodide of potassium to the cut surface of the cortical substance, we soon see bright reddish brown points and branching lines appear, standing out in lively contrast against the pale background, which scarcely shows any tinge of yellow from the iodine. These mark the Malpighian tufts and the arteries with their branches, which have undergone amyloid degeneration.

Grainger Stewart distinguishes three stages of amyloid degeneration. Besides the first beginnings of the disease, which scarcely cause any change that the naked eye can notice, he takes as his second stage the above described state of *swelling;* and lastly distinguishes a third stage of atrophy, which may advance up to a high grade of *contraction* of the kidneys. I do not, indeed, doubt that, under the influence of amyloid degeneration alone, an anæmic necrosis and destruction of the epithelium within the tubuli may take place, and in this way a considerable reduction be effected in the size of the previously swollen organ; but, from all that I have hitherto seen myself, I must declare my adherence to the opinion entertained by Virchow and Klebs, that the cases thus described by Grainger Stewart were not simple cases of amyloid kidney, but were cases of amyloid degeneration, with simultaneous, or, more likely, with a previously existing contracting disease (the genuine or secondary contracting process). I, at least, have never seen a kidney that was smaller than normal which was the seat of amyloid degeneration alone, and did not present at the same time distinct evidences of connective tissue hyperplasia.

The microscopic study of the development of this degeneration shows that *it first affects the renal vessels, and always begins by attacking the vascular tufts of the glomeruli*, the walls of which first exhibit the degenerative change in distinct points, and afterwards undergo the vitreous conversion throughout their extent. At a later date the afferent arteries degenerate, more rarely the efferent ones too, and the capillaries of the cortical substance. At the same time degeneration of the arteriolæ rectæ generally sets in, which subsequently often becomes excessive. A similar change may subsequently affect the tunica propria and epithelium of the tubuli uriniferi.[1]

The peculiar polish and the enlargement of the Malpighian vascular tufts are the first things that attract our attention, when fine sections, cut from kidneys which have undergone amyloid degeneration, are placed under the microscope. The whole mass of capillary coils appears larger and more voluminous than is normal. The smaller arteries of the kidney likewise appear more or less enlarged, and their walls are increased in thickness. The uriniferous tubes, with their epithelial linings and the interstitial connective tissue, often evince no sort of alteration whatever. According to Klebs, the amyloid degeneration only in rare instances attacks the tunica propria and epithelium of the uriniferous tubules. Grainger Stewart found the basement membrane often thickened and polished like wax, and the epithelium swollen and obscurely translucent; but the basement membrane in only a few instances showed the iodine reaction, and the epithelium never. Granular opacity and fatty degeneration are, however, to be observed in the epithelium.

---

[1] *Klebs*, l. c. S. 672.

The reaction with iodine and other reagents of the tissues that are the seat of amyloid degeneration is seen in the most perfect manner if one makes a fine section of the kidney with a double knife, and then, after carefully washing the specimen, touches this with the iodine solution, and examines it under the microscope. The Malpighian bodies then appear as reddish brown tufts, and the arterial stems which support them look like cylinders similarly colored in the midst of surrounding tissues of a simple pale yellow color. A drop of concentrated sulphuric acid added to a preparation thus treated immediately converts the reddish brown structures to blue. By this test alone one may at once convince oneself that all the Malpighian tufts, and all the branches of smaller arteries of the kidney are not affected at the same time or in the same degree. It is not every corpuscle or arterial stem that will assume the red-brown color with iodine; many, on the contrary, remain merely pale yellow, like the surrounding parts, and do not change their hue, even upon the addition of the sulphuric acid; while side by side with them lie glomeruli and vessels which exhibit the most vivid reaction.

It is possible, too, to study this condition still better by selecting kidneys for microscopical examination which have already been injected through the renal artery with a blue injection fluid, as my colleague Heller has had the goodness to show me. In preparations thus made it is easy to satisfy oneself that the amyloid degeneration does not merely alter the thickness and the aspect of the walls of the affected vessels, but that the higher grades of degeneration, by dint of the swelling of the walls, may completely close the calibre of the enlarged capillaries of the glomeruli. Into these glomeruli the injection fluid will not pass at all; they stand colorless beside those that are still permeable and are filled and colored by the injection. If such a preparation is dipped into the iodine solution, the unfilled glomeruli assume a distinct reddish brown color, while those filled with the blue do not alter their hue. Thus we encounter glomeruli which conduct themselves quite differently beside each other in the same field of vision. Certain glomeruli, too, present themselves, in which some capillaries have taken the injection mass, and remain uncolored with iodine, while other loops in the same tuft are empty, and offer a distinct iodine reaction.

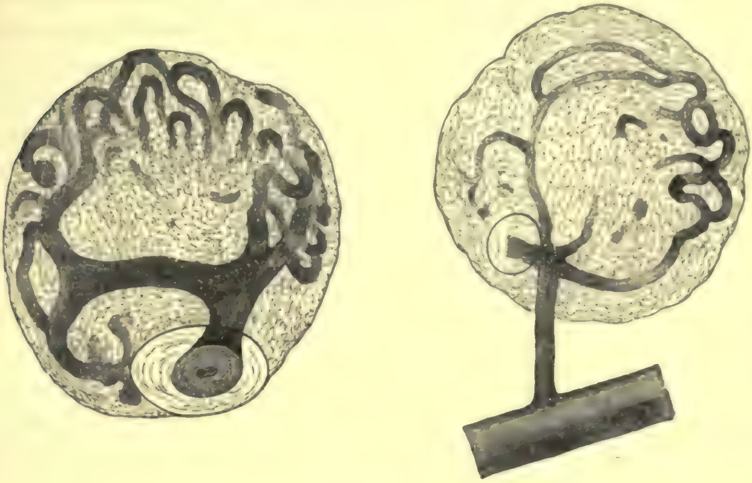


FIG. 14.

Injected glomeruli from amyloid kidneys. The shaded loops have received the injection fluid into them. (Heller.)

(Compare the accompanying Fig. 14, which is drawn from a preparation made by Heller.)

I have never yet made a post-mortem examination in which amyloid degeneration of the kidneys proved to be the only essential pathological condition present.

To begin with, I have, in every case, seen the same degeneration in some one or more of the other abdominal organs. It is well-known that the supra-renal capsules, above every other organ, almost invariably show this degeneration, if it is present in any organ of the body.

When the kidney is the seat of amyloid degeneration, the *spleen is almost always more or less involved in the same process; the liver more rarely so.* As to the state of the *lymphatic glands*, I have unfortunately not paid attention enough to them.

Degeneration of the *blood-vessels of the intestinal* mucous membrane is certainly a very common condition with amyloid disease of the kidney, and extensive ulcerative destruction of the mucous membrane is by no means a rare consequence thereof.

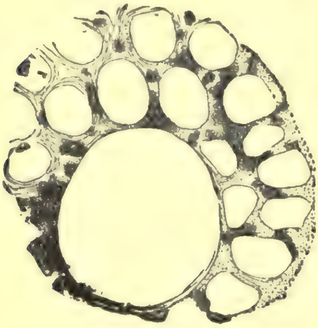


FIG. 15.

Collection of fat-globules in the intertubular spaces. Section made from a kidney in which amyloid affection of the vessels complicated parenchymatous nephritis. Magnified 150 diameters. (Colberg.)

The bodies of such persons are in the majority of instances dropsical. Anasarca may exist as well as dropsy of the various serous cavities. It has already been stated that the œdema of the skin is usually confined to the lower extremities, the genitalia, and the skin of the abdomen, and that the abdominal cavity is by far the most frequent serous cavity to contain dropsical effusions. *Most of these bodies are in an advanced stage of marasmus, but by no means all of them.*

In by far the larger proportion of cases on post-mortem examination we discover gross pathological changes in one or another of the internal viscera which stand in causal relationship to the renal affection. Taken in the order of frequency, *destructive ulcerations of the lungs and of the intestines by chronic tuberculosis* stand foremost in the list; then come *ulcerative (carious) diseases of the bones and joints;* next follow the *destructive ulcerations of the skin and of bone produced by syphilis;* and afterwards *extensive lesions of the skin effected by scrofulous and other ulcers.*

Every *chronic process of ulceration upon the mucous membrane of the intestines*, be its exact nature what it may, appears to me to deserve especial mention here; the same is true also of the follicular ulcer of the large intestine; and scarcely less important are the ulcers that form upon the mucous membrane of the urinary passages, in the pelvis of the kidneys, in the ureters and the bladder, all which I have found in the dead body, together with amyloid degeneration of the kidneys.

That amyloid disease of the kidneys is extraordinarily often complicated with *parenchymatous nephritis* I have already stated repeatedly; it is much less frequently associated with *contracting kidney*. But to this complication I shall come back in the appendix to this chapter (compare, however, Fig. 15 upon the preceding page).

As sequelæ of the amyloid affection of the kidney, I must make mention of the relatively frequent occurrence of thrombosis of one or both renal veins—a thing which I have met with several times in the bodies of extremely wasted individuals.

Loose infiltrations of the tissue of the lungs and purulent effusions into the serous sacs of the body, most frequently into the abdominal cavity, are not very rare conditions at a post-mortem. In one case I found a fresh purulent effusion into the left knee-joint.

I have only once seen *hypertrophy of the left ventricle* in the dead body, together with extreme amyloid disease of the spleen and kidney; this was in the person of the Swede so often mentioned by me, who was of quite herculean build. On the other hand, *I have repeatedly found the heart small and atrophied.*

Analysis of the Symptoms.

From our description of the course of the disease and of its pathological appearances, it follows that very many of the symptoms observed in patients afflicted with amyloid degeneration of the kidneys are either wholly independent of, or only in part referable to, the renal malady.

If we, therefore, first confine our attention to the disturbances which the renal functions suffer by reason of the degeneration of vessels spoken of, a great difficulty presents itself in accounting for them; for the alteration in the renal activity of the kidneys varies so greatly in different instances that it appears out of the question to assign the same cause to deviations from the normal secretion which are diametrically opposed to each other.

In those cases in which the kidneys persistently secrete large *quantities of watery and but slightly albuminous urine, we are perhaps right in assuming that the secretion takes place under conditions akin to those which prevail in simple contracting kidney, and that the secretion furnished must also be like that supplied by contracting kidneys.* Just as in simple contraction, so in amyloid degeneration of the kidneys, a large part of the vascular secreting apparatus is impermeable to the blood-stream. The necessary consequence is that the blood-pressure rises correspondingly high in the parts which remain pervious. More fluid will, therefore, filter, and that with increased rapidity, through the vessels which still perform their functions. This filtrate is from the first abnormally aqueous, because it emanates from a blood-serum which is, as a rule, abnormally watery. Given a certain amount of blood-pressure, and it will also contain albumen.

From very weak solutions of albumen this substance transudes through the pores of the filter under less pressure than it does from more concentrated solutions. Upon its passage through the uriniferous tubules, which it traverses with great rapidity, this original filtrate from the glomeruli which still retain their activity will undergo no great changes, partly because, the blood-serum flowing in the capillaries being itself highly watery, the conditions for osmotic interchanges are but feeble and operate with very slight intensity, and partly because, in most cases of amyloid disease, the diminished tissue metamorphosis furnishes but a scanty supply of specific urinary constituents to the renal cells for separation. Hence the low specific gravity of abundant urine.

All these theories, however, fall to the ground in the face of those cases in which abnormally scanty amounts of urine are secreted of quite abnormally high specific gravity and extremely loaded with albumen. Here there is but one thing, I think, that we can positively assert, namely, that the walls of the secreting vessels in these cases must be altered in some way, perhaps by the amyloid degeneration itself, and in a manner which renders them pervious to serum-albumen. In such cases the urine resembles in many respects that furnished by kidneys in a state of chronic parenchymatous inflammation. I was for a long while of the opinion that in all the cases of this sort that I had previously observed, the amyloid disease might have been complicated with this form of nephritis without my having recognized it ; but since that time I have been convinced by a series of clinical observations and close anatomical investigations, that this same thick, highly albuminous urine may arise in perfectly pure cases of amyloid kidney. The character of the blood-serum has not such a distinct influence upon the amount and concentration of the urine as to account for the great differences in quality of the urine furnished in different instances of amyloid disease of the kidney. The scanty secretion of a heavy, highly albuminous urine occurs in dropsical individuals (those, therefore, whose blood-serum is very watery) as well as in those who are still quite robust. The unequal vigor of the heart's action, too, need not be considered here ; for, among all my patients, one only had any hypertrophy of the left ventricle. He passed plenty of urine, but it was invariably of very high specific gravity, and contained a large amount of albumen.

There remains, therefore, but one explanation open to our acceptance, viz., that the amyloid degeneration, so long as the vessel remains pervious to the blood-stream, renders the walls of the vessels generally permeable to albumen, and that, therefore, all the albumen in the urine passes through the walls of the diseased vessels, and is not furnished by the sound vessels, as was claimed by our first hypothesis. The dissimilar amounts in which albumen appears in the different urines might then depend upon the degree and extent of the degeneration of the vessels ; but, to decide this question, more exact clinical and pathological investigations will be requisite.

But even if this question of the real source of the albumen in amyloid disease of the kidney were decided, still the other question of the cause of the great dissimilarity in the amounts of urine secreted under conditions apparently so identical would not be settled. Here too, seeing that the disease of the glomeruli imposes great obstacles to the circulation of the blood, and that the secretion of urine must thus be interfered with by extensive spread of the degeneration, we might, for the present, explain the phenomenon in question by supposing that these variations in amount depended on the grade and extent of the disease, leaving the final decision of the matter to some future period.

For the present, at all events, it is impossible to determine what influence is exercised on the aggregate functions of the kidneys by the various factors which come into play, among which we may mention the limitation of the blood-channels by the occlusion of so many vessels, the altered permeability of a large portion of the secreting vessels, the altered composition of the blood, etc.

The elimination of the specific urinary constituents, and especially of urea, by the kidneys, appears to be scarcely at all affected by amyloid degeneration of the secreting vessels. So long as secretion continues to take place at all, the urine furnished by amyloid kidneys may contain a high percentage of urea, as well as large daily amounts of this substance. In cases where the autopsy has shown extreme amyloid degeneration of the kidneys, I have repeatedly witnessed, but a few weeks before death, daily excretions of urea such as exceeded the ordinary physiological standard. Nor need this surprise us, if we consider that the specific glandular cells in amyloid kidneys are often perfectly normal in structure, and are present in undiminished numbers. When, nevertheless, in particular instances, both the percentage and total amount of urea excreted fall far below what is normal, this is very rarely due to secondary degeneration of the renal epithelium—an event which seldom happens to any considerable extent,—but is attributable to general marasmus and arrest of tissue metamorphosis throughout the system at large.

It is for this reason that uræmic symptoms are so exceedingly rare in amyloid disease of the kidney, not even occurring when, toward the end of life, the excretion of specific urinary constituents falls to minimum rates. These small quantities may, nevertheless, correspond to the diminished production.

The only case of *uræmic convulsions* I have met with, occurring in amyloid degeneration of the kidney, is the following:

Case XXX.—Claus Woebs, a carpenter, thirty-five years of age, first came into our Medical Clinic on the 8th of August, 1868. He was complaining of pain over his abdomen and of general irritability. Ever since 1865 he had noticed some gradual diminution of his strength, enough to make his occupation irksome to him. He claimed that he had never had syphilis. The first notes taken merely show that the patient looked very wretched, and that his urine contained *much albumen*, and had a specific gravity of 1026.

Since he would not enter the Clinic, the patient was advised to present himself from time to time to be seen.

Condition, Oct. 9, 1868: Patient looked pale. Examination of the lungs gave negative results. *The amount of urine passed in twenty-four hours was* 900 *c.c., sp. gr.* 1022, containing a considerable amount of albumen. Ordered decoction of cinchona.

Condition, Dec. 2: Latterly the patient had had attacks of headache, and had complained of pains on the right side of his abdomen. Appetite good. According to his own account, he weighed 144 pounds, with his clothes on. *Amount of urine in twenty four hours*, 1,325 *c.c., sp. gr.* 1021. Ordered iron pills.

Condition, Jan. 1, 1869: In the cæcal region, upon the left side, the presence of a tumor was ascertained. It was about the size of an apple, sensitive to pressure, and but very slightly movable. After this date the tumor was felt at each subsequent examination. The amount of urine in the twenty-four hours was 1.725 c.c., sp. gr. 1019. Upon a mean of fifteen analyses instituted between August, 1868, and January, 1869, Woebs was ascertained to have excreted a daily average of 900 c.c. of urine, containing 24.6 grms. of urea and 5.5 grms. of albumen.

Condition, Feb. 21, 1869: Patient was very anæmic. Appetite bad. *For five weeks he had had profuse diarrhœa*, associated with some tenesmus, stools apparently containing *pus*. Patient stated that during the winter, for the past three years, he had had a similar attack of diarrhœa. Ordered opium and syrup of the iodide of iron. Urine for the twenty-four hours, 1,150 c.c., sp. gr., 1015; much albumen.

Condition, February 28, 1869: Slight dropsy of the lower extremities; diarrhœa just as before, associated with abdominal pain; loss of appetite; cough; examination of the lungs showed catarrhal affection of the apices and doubtful respiratory sounds; ordered Potio Riveri cum opio. The twenty-four hours' urine amounted to 675 c.c., sp. gr. 1018; much albumen; urine somewhat cloudy.

Condition, March 8, 1869: Diarrhœa had somewhat abated; patient looked very anæmic and feeble; had suffered from violent hiccough for last twenty-four hours. Patient was now admitted into the Clinic.

State on admission: A most highly cachectic-looking man; voice feeble; complained of pain in the region of the larynx, especially on coughing or swallowing; constant, exceedingly troublesome hiccough; no dropsical swellings; frequent stools of greenish brown color, fetid and of a thin fluid consistency. Patient coughed pretty frequently, and expectorated some coffee-grounds sputa; ordered red wine and decoction of cinchona bark. Passed very little urine.

March 10th.—Patient does not any longer appear quite sensible; the motions of the bowels are frequent and the hiccough continues.

Evening.—Appears nearly insensible, passes everything under him; has had repeated attacks of general convulsions, during which bloody froth exudes from his mouth.

March 11th.—Death at one A.M. this morning.

Post-mortem examination.—Lungs. The surfaces of both lungs showed large cicatricial contractions, and were scattered over with numerous nodules of a white color, and as large as hazel-nuts. These nodules were of a peculiar elastic consistency, and were separated from the air-containing lung tissue round about them by a remarkably distinct line of demarcation. Upon section of these nodules distinct ramifications of blood-vessels were to be distinguished, filled with blood. Under the microscope it was apparent that these nodules consisted of thick masses of the finest cells, imbedded in a homogeneous matrix. I believed that they should be reckoned as gummy tumors.

Heart, nothing noticeable. *Spleen*, rather enlarged, remarkably hard in consistency, and coarsely granular upon section.

Left kidney rather enlarged, pale, its capsule separating easily. Upon section the cortical substance proved to be broader than normal, and remarkably pale, whereas the pyramidal cones appeared of a bright reddish brown.

Right kidney relatively rather larger than the left, but exhibiting the same appearances, with the addition of some catarrhal swelling of its pelvis. The *lymph glands* situated round the entrance of the portal vein into the liver and between the folds of the mesentery were swollen, had a medullary aspect upon section, and were pale and anæmic.

The intestines from the rectum up to the middle of the ileum showed a large number of quite peculiar ulcers. They were broad losses of substance of the mucous membrane, encircling the entire tube of the intestine like a girdle, the muscular coat of the bowel appearing bare and polished at the base of the ulcers. The edges of the sores were smooth, as if punched out, and were firmly fixed down to the base of the ulcers. In many places the lymph vessels round about these ulcers appeared distended with white milk-like contents in long tracts, and here and there they were dilated into ampulliform sacs looking like a string of pearls. This ulcerative destruction reached its greatest extent in the mucous membrane of the cæcum. Here the entire intestine was stripped of its lining membrane, and the external layers of its walls were so thickened by cicatricial tissue as to have caused the tumor, the size of an apple, which could be felt during life. Through the cicatricial contraction of this hard callous mass the calibre of the cæcum was considerably narrowed, particularly the point of entrance of the ileum, the ileo-cæcal valve being contracted to such a degree that it scarcely admitted the passage of one's little finger.

This destruction of the mucous membrane of the intestine I believe I am perfectly justified in referring to a past syphilitic process. The ulcers were totally different in all their features from tuberculous ulcers, just as the nodules found in the lungs were quite unlike large caseous tubercles; indeed, the difference was clearly recognizable, even to the naked eye, by the blood-vessels which ramified through them still containing blood.

It is a well-known fact that many patients deny previous syphilitic infection, even when they know better.

The relation of the renal affection to the general nutrition and to the occurrence of dropsy has been already stated above, where it was also pointed out that the loss of albumen in the urine, and the occasional diminution of the excretion of water through the kidneys, favored the progress of hydræmia, though they are not its sole promoters, and are certainly rarely its essential cause.

Prognosis.

The prognosis of amyloid disease of the kidneys is, in the first place, entirely dependent on the original fundamental malady, and since in most cases the renal degeneration arises from a condition which is absolutely hopeless, the prognosis is certainly, *as a rule, positively unfavorable.* We are, indeed, undoubtedly justified in the assertion that the addition of this renal degeneration to the pre-existing state of disease must not merely be considered as an unfavorable omen, showing that the general nutrition is gravely involved and that the prolongation of life is seriously threatened, but as a circumstance which often hastens the fatal event in a very decided manner. This is so much more surely the case, because at the same time with the kidneys, or even before them, the spleen and other organs are attacked with the same degeneration, and have their functions disordered. Although up to the present time we are but little acquainted with the significance of affections of the spleen in the natural history of vital functions, still experience teaches us that disease of this organ exerts a decidedly prejudicial influence thereon.

What prognosis are we to give, however, in those rarer instances in which the amyloid disease of the above-named organs occurs without any previous apparent cause? Does the general verdict hold good for them, too, which has been passed upon the others, to the effect that the amyloid affection is, of all the diffuse diseases of the kidney, the one whose prognosis is most unfavorable? To this last question I think I may positively answer "*No*," and appeal to the experience already detailed above to corroborate me.

But if a more favorable prognosis is admissible in these cases, then, in other cases too, under some circumstances, we must be able to give a better prognosis than is generally regarded as justifiable in the opinion of physicians; as, for example, where it may be possible to remove the cause which, in any given instance, may have provoked the amyloid degeneration, and so do away with its injurious action both upon the kidneys and upon other organs.

As to the possibility of a retrogressive metamorphosis taking place in tissues already involved in amyloid degeneration, I will not venture to hazard any conjecture here, but shall only call attention once more to the fact that, in each of the two cases related above, the spleen, which previously had been greatly swollen, and, in one instance, the liver also, were again reduced nearly to their normal volumes, and that, in both instances, the albuminuria, which had been pretty considerable, and, besides, had lasted for some years, disappeared completely.

This does not, however, prove that the walls of the blood-vessels, and other tissues that have been attacked with amyloid degeneration, can resume their previous normal condition again. In respect to the kidney, specially, I have pointed out that, even in the very advanced cases of this affection, all the vessels of the organ are not affected. It appears possible, at all events, that, in the cases that recover, the parts which have escaped degeneration, accommodating themselves to the necessity, may suffice to fulfil the requisite functions of the kidney. This is the more likely, as experience teaches us that, after the loss of one entire kidney in an adult man, the organ that is left is equal to meeting the entire demands of the economy. A complete answer, however, to the question here mooted must be deferred until further investigations have been instituted.

In cases in which the fundamental malady that has provoked the amyloid disease cannot, in itself, be considered a sufficient cause of death, as, for example, in many instances of syphilis, the amyloid affection may last for years before the patients succumb to it; and in these cases it appears, according to my own experience, that the simultaneous affection of the spleen, the liver, and the intestines, is much more responsible for the final fatal termination than is the renal disease. For, as I have already above remarked, the affected kidneys often continue to excrete a sufficient amount of urea up to a short time before death. In saying this, however, I do not intend, by any means, to underrate the damaging effect produced by the loss of albumen due to the renal affection.

As a rule, in these cases, the amyloid disease leads on to death by the following succession of symptoms, occurring in the order mentioned: anæmia, albuminuria, dropsy, diarrhœa. The sudden occurrence of symptoms of collapse often precedes death with equally sudden reduction, or else complete suppression, of the previously profuse secretion of urine. The following case took just this, so to speak, normal course:

Case XXXI.—Mrs. A. E., thirty-two years of age, was taken ill with the symptoms of parametritis, four days after her confinement in July, 1867, the delivery having passed over easily. The parametritis, after a full month, or rather more, went on to the formation of a pretty considerable exudation into the abdominal cavity. Later on, this perforated into the bowel, and was succeeded by occasional very abundant discharges of pus per rectum. Under symptoms of continued fever and persistent pain in her abdomen the patient soon became extremely emaciated. In May of 1868 she noticed that wind passed with the urine, and she was constantly constrained to micturate. Neither pus nor fæces had ever been mixed with her urine. She now determined to enter the hospital.

The abdomen, which was examined as the patient lay on her back, was pretty well distended; it gave clear, tympanitic resonance on percussion down to the navel, but was dull below this, being dullest of all on both sides, over Poupart's ligament. At this point hard masses of exudation could be felt, while the abdomen was very tender upon pressure.

By catheterism, which was effected with some difficulty, on account of the extreme sensibility of the urethra, a moderate quantity of light colored, somewhat turbid urine was drawn off, but after this had passed a puff of gas escaped from the bladder with a loud noise.

The urine of the twenty-four hours amounted to 1,060 c.c., sp. gr., 1015.5. There was a trace of albumen in the urine; reaction acid; a moderate quantity of pus corpuscles was present in the sediment.

In the further progress of the disease copious diarrhœa set in, scanty masses of pus being often mixed with the dejections. The abdominal distention remained little altered, the pains and the constant desire to micturate continuing, with small intermissions, to be the perpetual cause of the patient's complaining. At the end of January, 1869, there was a marked change for the worse in her condition. *The* *lower extremities became anasarcous, and the œdema gradually extended over the rest of the body.*

Coinciding with this deterioration in her state there occurred *a remarkable alteration in the character of the urine;* up to this date the daily analysis of her water had shown that it did not contain more albumen than could be referred to the amount of pus that was mixed with it, but the examination instituted on Feb. 1, 1869, showed the presence of a considerable amount of albumen (0.5 per cent.), whereas the sediment contained comparatively little pus. From this time forth the daily average amount of urine was 1,580 c.c.; its sp. gr. fluctuated between 1004 and 1015, while the percentage of albumen was between 0.19 and 0.81. In the sediment were seen *some narrow, extremely pale casts* in a state of fatty degeneration, besides colorless blood-corpuscles and epithelium from the urinary passages.

With the general increase of the anasarca and the violent diarrhœa, the patient's powers rapidly decreased, and death ensued on the 28th of March, with symptoms of complete collapse, after the urinary secretion, which had been very abundant shortly before, had ceased almost altogether during the last few days.

The autopsy proved the existence of a communication between the bladder and the colon, as also a perforation of the cæcum, with extensive ulcerations of the bowel. As to the rest of the post-mortem record, the following extracts only need be given:

Heart small, and strikingly pale. The walls of both ventricles, especially the right, very thin.

Spleen adherent to the parietal wall of the abdomen by the whole of its surface; nearly twice its normal size, and perfectly stiff; capsule bluish, with some large, tendinous patches. On section its substance was polished, firm, and the color of red wine. It became brown upon the addition of iodine solution, and bluish black spots became apparent when sulphuric acid was added.

Left Kidney considerably enlarged; easily shelled out of its capsule; its surface of a pale gray color, showing numberless little vortices. Length = 13 cms.; breadth = 5 cms.; thickness = 6 cms. Upon section, the cortical portion exceeded the pyramidal. The color of the former was grayish red, dotted here and there with yellow spots, and traversed with some large blood-vessels. The pyramidal portions were also very pale, and presented alternate streakings of red and pale gray lines. Pelvis of the kidney reddened, and in an advanced stage of catarrh, the mucous membrane being highly swollen.

Right Kidney adhering to the descending portion of the duodenum. Capsule strips off easily. Length, 11.5 cms.; breadth, 5 cms.; thickness, 5 cms. Color of both external and cut surfaces the same as that of the left kidney; but, unlike this, its pelvis presented no trace of any catarrhal swelling, and its mucous membrane was perfectly pale.

Liver remarkably small, rather globular, moderately firm, and containing very little blood. There was moderate distention of the vena centralis of the individual lobules.

Diagnosis.

The diagnosis of amyloid degeneration of the kidney is essentially etiological, and must be so from the very nature of things, since the symptoms provoked solely by the renal malady are such as may be occasioned by other forms of kidney disease. It is only from our knowledge of the fact that amyloid degeneration of the kidneys frequently occurs under certain circumstances (given above), that it is possible by careful examinations of the urine to recognize this affection at its commencement, since *we arrive at its recognition from no other symptom except the presence of albumen in the urine.* But chronic parenchymatous inflammation of the kidneys, which is likewise associated with albuminuria, arises under precisely the same circumstances; and thus, in some instances, extraordinary difficulty is experienced in making a positive diagnosis—a difficulty which is not always removed by continued examinations of the urine.

In both cases the amount of urine passed per diem may be small, its specific gravity abnormally high, and the quantity of albumen contained in it exceedingly large. The following results of urine analysis in the two cases may serve as a guide to diagnosis, although none of them can lay claim to universal applicability:

In *amyloid disease* the urine is scanty, clear, and rarely forms a sediment; it is darker colored, contains very few casts, and these mostly of the hyaline variety; it scarcely ever contains red blood-corpuscles. The amounts passed and the characters of the urine are subject to frequent and considerable variation.

In *chronic parenchymatous nephritis* the urine is scanty, is invariably more or less turbid, and usually deposits a considerable amount of sediment; it is rather of a dirty than a dark color, and usually contains an abundance of casts of every variety, and not uncommonly either a few scattered or else an abundance of red blood-cells. The amounts passed and the characters of the urine, as a rule, do not vary much, and then only at longer intervals of time.

It would prove of great diagnostic value if it should turn out, as Senator thinks, that the urine in amyloid degeneration of the kidney is especially distinguished from other albuminous urines by the large amount of globulin contained in it.

Amyloid degeneration of the kidney, as well as chronic parenchymatous nephritis, is usually associated with dropsy, the latter certainly more often than the former—indeed, almost invariably. Roberts adduces the difference in the mode of extension of the dropsical effusions over different parts of the body as a valuable means of distinguishing between them. He states that in amyloid disease the dropsy confines itself to the lower extremities and the abdominal cavity, whereas in nephritis, as a rule, the anasarca is general, and effusions take place into all serous sacs indifferently. Among the greater number of my own cases of amyloid kidney I have certainly found the opinion of Roberts as to the limitation of the dropsy confirmed, although it has not held good for all.

The demonstration of *swelling of the spleen, and sometimes of the liver too*, is a point of great importance in the differential diagnosis of the two kidney affections under consideration. Enlargement of the liver, however, is altogether absent in by far the larger number of cases of amyloid disease, *and amyloid livers, too, may even be smaller than normal ones.* This, however, is far less apt to be the case in spleens which are the seat of amyloid degeneration. But I have found that even when the spleen is considerably enlarged by amyloid disease, the area of dulness is by no means correspondingly increased, because by far the larger half of the enlarged spleen in such cases, as the post-mortems prove, lies within the vault of the diaphragm and removed from the wall of the ribs. This perhaps arises from the so frequent occurrence of abdominal distentions in amyloid disease by gaseous accumulations in the intestines or from ascites. It rarely happens that the splenic tumor is so large that one can feel it beneath the left costal arch. *When the ascites is excessive, percussion can give no information concerning the size of the spleen.*

Grainger Stewart describes a quite peculiar color of the face, and especially of the eyelids, as a feature characteristic of amyloid disease, this being due to the deposition of dark pigment in the tissue elements of the skin. I have certainly found that the faces of many of my patients, but by no means of all, presented a dirty earthy color—a complexion quite unlike that simple pallor which occurs in persons with nephritis. But I never remarked any localized pigmentation upon the eyelids, and consider the *muddy complexion in question as the expression of the fundamental cachexia rather than of the secondary amyloid disease.* I am equally unable to attach diagnostic value to another symptom given by the above-named English observer—namely, a circumscribed red patch upon the cheeks from ektasis of the small cutaneous veins. This circumscribed dark red patch I have certainly seen in some of my cases, but it is altogether absent in by far the larger number of them. When it occurs, its cause lies in mechanical disturbances of circulation, which in two of my own cases were due to cirrhosis of the lungs with bronchiektasis.

If the amyloid degeneration of the kidney is associated with polyuria, as it most commonly is, the affection becomes scarcely less difficult to distinguish from genuine contracting kidney, since in cases of this nature the renal secretion, both in quantity and quality, may be exactly like that which is furnished in the last-named disease.

Then, once again, a review of the etiological conditions, the more frequent appearance of dropsy, the evidence of a splenic tumor, and, above all else, the absence of every symptom indicative of cardiac hypertrophy, would speak decidedly in favor of the existence of amyloid degeneration, and against that of simple renal atrophy.

Still, cases do occur—those, for instance, in which no definite etiological condition is apparent—where, even when every circumstance above referred to has been carefully considered, an error of diagnosis can scarcely be avoided. Mistakes of this kind have frequently occurred to me in the case of patients who were brought nearly moribund into the hospital.

Finally, I must once more insist upon the fact that amyloid degeneration of the kidney complicates both chronic nephritis (a very common combination) and contracting kidney. In such combinations of disease, I, for my part, must at present decline hazarding a positive diagnosis.

Treatment.

From what has been said on the subject of prognosis, it will be apparent how gloomy is the prospect of treatment in amyloid renal disease; but, for all this, I think, as therapeutists, we are not to stand by with folded hands. Our endeavors to save the lives of phthisical patients whose kidneys are already excreting albuminous urine, will, of course, be in vain; but it is certainly an open question whether many a life might not be saved if we were resolved to sacrifice a diseased member at the proper time, that is, as soon as the first symptoms of the renal disease manifested themselves. For myself, not only should I consent to it at once, but, under certain circumstances, should consider myself in duty bound to have it done.

In short, preventive treatment is certainly at present the most powerful weapon against this destructive process; *and herein I include, above all other things, a more thorough, radical treatment of syphilis, that scourge of our age, than is at present ordinarily practised.* If we permit our syphilitic patients to give up treatment as soon as their sores are healed, their exanthematous eruptions have faded, and their condylomata have disappeared, it is not to be wondered at that a relapse should speedily ensue, and that, after undergoing frequent and repeated half-cures, our patients should at last fall into a condition of incurable sickness and cachexia. *I never let my own syphilitic cases give up their mercurial treatment until all the swollen lymphatic glands have lost their swelling, even if this takes many months.* I have had every reason to be satisfied with the results of my practice, so far as the frequency of relapses is concerned, and I believe my patients have even more reason to be satisfied, although they have often enough grumbled at my protracted course of treatment. But in this respect I am still far behind the requirements recently advocated by our old master, Ricord, in the paper which he read at the Birmingham Congress; he requires each syphilitic patient who puts himself under his care to take iodide of mercury for at least six months, and iodide of potassium for six months longer.

Preventive measures, however, are not excluded even when the amyloid disease has already begun, for even then we may perhaps make up for lost time in drying up the fountain from which the mischief springs.

When full justice has been done to the *indicatio causalis*, then we may try to get directly at the amyloid disease that has broken out. The most various propositions for treatment have been advanced, corresponding to the different views held concerning the essential character of this degeneration. Dickinson, who (after Lecorché) considers that the amyloid degeneration is the result of a loss or waste of alkaline salts, which the body experiences in consequence of prolonged suppuration, orders carbonates or vegetable salts of the alkalies. Others pretend that the amyloid substance will melt down under the action of nitric acid, given either internally or in baths. But I am not aware that the desired result has been attained by the application of these remedies.

I shall, therefore, for the future have recourse to the same plan of treatment to which I believe I am indebted for the favorable issue of the cases which recovered under my hands, and which I have detailed above. I shall continue to order iodide of potassium to these patients, although I do not know how this medicament acts upon the diseased walls of the vessels; and shall endeavor, at the same time, to support the general nutrition by preparations of iron, vigorous diet (meat and milk), and good wine, and to maintain sufficient action of the skin by baths.

Besides this, as a matter of course, the indications derived from the symptoms which each particular case affords must receive proper attention, according to the directions laid down in the previous chapters of this work.

Appendix.

The Complications of Amyloid Degeneration of the Kidney with Chronic Parenchymatous Nephritis and with Contracting Kidney.

It has already often been intimated that in the dead body amyloid degeneration of the vessels of the kidneys, more or less widespread, is very often found complicated with chronic parenchymatous nephritis, and far more rarely with contraction of the kidneys.

With regard to the complication of amyloid degeneration with chronic parenchymatous nephritis, I must first remark that the combination is one that I have met with almost exclusively in the bodies of those who were the victims of inveterate syphilis. In such subjects I have found, besides the renal disease, syphilitic destructions of the skin and bones, and gummata of the liver, as well as syphilitic ulcerations of the intestines.

In such cases, therefore, nothing stands in the way of our assuming that both renal processes, being derived from the selfsame cause, may have begun and have been developed simultaneously ; at any rate, that they are both effects of the same cause, and do not stand in the relation of cause and effect to one another. But I must here remark that the renal secretion in such cases has been altogether like that of chronic nephritis, especially in those instances observed from the commencement of the renal malady, that is, of the albuminuria.

The hypothesis of nephritis complicated with amyloid degeneration could, therefore, only have been based upon etiological grounds.

The case is quite different in the complication of amyloid degeneration of the vessels with connective-tissue hyperplasia, and contraction of the kidney. Here it has been assumed that the amyloid degeneration of the vessels is the primary event, and the anæmic necrosis and wasting with absorption of the renal epithelium, with final atrophy of the entire organs, are the secondary result or sequence of the affection of the blood-vessels. This view seems to me to be opposed by the fact of the unmistakable increase in bulk of the interstitial connective tissue which occurs even in these cases, and the hypertrophy of the left ventricle which occasionally accompanies it. I should rather advocate the opinion that in these cases the gradual cachexia produced by genuine contraction of the kidney was the primary mischief and the cause of the secondary amyloid degeneration of the vessels of the kidneys, as well as of those of other parts.

In such cases as these I consider that a diagnosis is only possible when, in addition to hypertrophy of the heart, a distinct splenic tumor has developed itself; or when some etiological reasons have rendered the amyloid affection probable; yet these things are especially liable to be absent in cases thus complicated.

The urinary secretion behaves just as in simple contracting kidney, and an examination, therefore, of the urine will not prove of any value in assisting us towards ascertaining the existence of this complication.

DISEASES OF THE KIDNEYS,

INCLUDING

THE AFFECTIONS OF THE RENAL PELVES AND THE URETERS.

EBSTEIN.

DISEASES OF THE KIDNEYS,

TOGETHER WITH

THE AFFECTIONS OF THE PELVES OF THE KIDNEYS AND THE URETERS.

Troja, Krankheiten der Nieren. Deutsch. Leipzig. 1788.—*Walter*, Einige Krankheiten der Nieren. Berlin. 1800.—*Baillie*, A series of engravings. II. edit. London. 1812.—*G. Koenig*, Praktische Abhandlungen über die Krankheiten der Niere. Leipzig. 1826.—*Lebert*, Traité d'anatomie pathologique générale et spéciale. Paris. 1857-1861.

The works upon diseases of the kidneys by *Rayer* (1839), *Johnson* (1852), *Vogel* (1865), *Rosenstein* (1870), *Roberts* (1872); upon pathol. anatomy of the kidneys, consult *Rokitansky* (III. Bd. 1861); *Foerster; Klebs* (3. Lieferung. 1870).

Complete information of the earlier literature is given by:

Naumann, Handbuch der med. Klinik. VI. 1836. Berlin.—*Canstatt*, Handbuch der med. Klinik. IV. 3. Abth. 1845. Erlangen.

Inflammations of the Kidney, of the Pelvis of the Kidney, and of the Perinephritic Tissues, with Termination in Suppuration.

Suppurative Nephritis. Renal Abscess.

LITERATURE.—*Voigtel*, Pathol. Anatomie. III. S. 185.—*Virchow*, Gesammelte Abhandlungen. S. 602.—*The same*, Ueber die Chlorose, etc. Beiträge zur Geburtshülfe u. Gynäkologie. I. Bd. 1872.—*Beckmann*, Verhandlg. d. phys. med. Gesellsch. IX. Bd.—*The same*, Virchow's Archiv, XIX.—*Kussmaul*, Beiträge zur Pathologie der Harnorgane. Würzb. medic. Zeitschr. 1864. S. 56.—*Siebert*, Diagnostik der Krankheiten des Unterleibes. 1855. S. 382.—*Todd*, Clinical lectures on certain diseases of the urinary organs. London, 1857.—*Leyden*, Rückenmarkskrankheiten. I. 1874.—*Cohnheim*, Embolische Processe. 1872.—*Heiberg*, Virchow's Archiv. LVI.—*Burkart*, Berl. klin. Wochenschrift. Nr. 13. 1874.—*Eisenlohr*, Berl. klin. Wochenschrift. Nr. 32. 1874; and numerous articles scattered throughout the literature, to which references are partly made in the text. Compare, also, the general literature mentioned above.

Introductory Remarks and History.

I consider in this chapter those forms of renal inflammation which lead to the formation of abscesses, and, for certain practical considerations, only the *traumatic*, *idiopathic*, *pyæmic*, and *metastatic* forms, reserving for subsequent description several other groups that properly come under this head.

As regards the *traumatic* form of renal inflammation, Celsus has already given an account of the symptoms which follow injuries of the kidneys. In fact, an abundant material concerning suppurative processes of the kidney in general is to be found accumulated in the older literature. Still, a great confusion in the prevailing ideas regarding renal suppurations has existed up to the most recent times, because the affections of the pelvis of the kidney and those of the kidney itself were not sharply separated, everything being promiscuously designated as renal abscess. Hence, there are very many cases of renal abscess recorded in the older literature which are of no scientific value whatever, because, in reality, they have reference far oftener to pyelitis and pyelonephritis than to true renal abscess. To Rayer belongs the honor of having thoroughly and sharply separated these various pathological processes. Virchow's views regarding *embolism* and *thrombosis* have cleared up the pathogenesis of an entire series of renal suppurations, and furnish a sound foundation for the theory of metastatic nephritis. As a result of the revolutionary change of views caused by Virchow's great discovery, many sought to attribute to emboli *all* the abscesses of the kidney occurring in the course of the complex symptoms that constitute pyæmia. Experience, however, has shown that this generalization is not justified by the facts. On the other hand, it has taught us how the production of truly metastatic renal inflammations is to be explained. Cohnheim's researches have also led the van in this direction. Recently a great rôle has been ascribed to bacteria in the pathogenesis of these metastatic abscesses, but further observations are required to show whether and how far this hypothesis is founded upon fact.

Etiology.

Inflammations of the kidney resulting in the formation of pus and of abscesses are excited by various causes, of which the following are of especial practical importance:

1. *The presence of foreign bodies in the renal tissues*, which mechanically irritate them and excite suppuration. Here belong, first of all, *renal concretions.* They are, without doubt, the most frequent cause of renal abscesses. For the sake of conformity I have postponed the description of this *nephritis suppurativa calculosa* to the chapter on nephrolithiasis. It did not seem to me advisable, for the sake of the plan, to describe separately affections that are alike in their etiology and their course of development.

2. *Injuries* which befall the kidneys in various ways. Here belong those suppurative inflammations resulting from gunshot, incised, or punctured wounds, and also those injuries of the kidney produced by falls, blows, or kicks, which are occasionally followed by suppurative inflammation. It is hardly necessary to state that only a small fraction of these injuries cause suppurative inflammation of the renal tissues. When, for example, the kidneys are injured by a fall from a considerable height, rupture of the organ or of its pelvis, if distended with urine, is the usual result. If rupture of the peritoneum occur at the same time, the injury proves rapidly fatal. If, on the other hand, the peritoneum be uninjured, the urine and blood are extravasated into the meshes of the perinephritic and retroperitoneal connective tissues, and excite inflammation and suppuration, with consecutive urinary fistulæ in many cases. This is frequently an exceedingly chronic affection, but one which is nevertheless susceptible of cure. In the present chapter only traumatic suppurative processes of the renal tissue itself will be considered.

3. *Chronic retention and decomposition of the urine.*—I have

described the renal suppurations arising in this manner under the head of pyelitis and pyelonephritis, because in these cases the renal pelvis and parenchyma are almost always simultaneously affected, and a separate description would involve too much repetition.

4. *The so-called pyæmic affections.*—Suppurative nephritis is rarely met with in pyæmia in comparison with suppurative processes in other localities. Abscesses of the kidneys are caused far more frequently by

5. *Metastasis through embolism of the smaller renal vessels with infecting substances.*—This may happen when portions of infected thrombi of the pulmonary veins are broken off and carried into the systemic arterial vessels. This, however, occurs very rarely. These metastatic renal abscesses occur almost exclusively in connection with malignant endocarditis sinistra, aortica, mitralis, or parietalis, which gives rise to the production of pernicious, specifically poisonous emboli, which occlude the finer renal vessels and produce miliary abscesses in the surrounding tissues.

The renal abscesses which come under the two last heads produce, as a rule, no characteristic modification of the clinical pictures of the original diseases. The same may be said of

6. Renal suppurations which are produced by extension of *inflammatory processes from the neighboring organs*, especially from the *perinephritic tissues*, *the psoas muscle*, *the liver*, etc. On the other hand,

7. The abscesses which are developed in the kidney *without any cause being discoverable, either intra vitam or post-mortem*, often become the objects of clinical observation. In how far exposure to cold, etc., must be regarded as causative factors in these cases cannot be satisfactorily determined with our present limited experience.

8. The misuse of active *diuretics*, especially of cantharides, causes inflammation of the kidneys, but we have not yet sufficient data to decide whether or not it must be classed with the causes of renal abscess. The same must be said of Rayer's supposition that nitre in large doses causes nephritis.

9. Earlier observers (Koenig) included suppressed hæmor-

rhoidal flux and excessive venery among the causes of renal inflammation. Accurate observations, however, are still wanting on this point.

Pathology.

Pathological Anatomy.

In traumatic nephritis the pathologico-anatomical lesions vary according as death ensues after a *shorter* or *longer* duration of the disease. In the former case the renal wound, with the changes in its vicinity, is sometimes still visible. Where no circumscribed breach of continuity has occurred, a diffuse alteration of the parenchyma is generally found. The organ is commonly enlarged, at times considerably so; the tissue is enormously vascular and spongy, the markings are more or less indistinct or have even completely disappeared, and here and there in the renal parenchyma smaller or larger collections of pus, and between them generally abundant extravasations of blood, are found. Occasionally the entire organ is saturated with a grayish white, purulent, turbid fluid. How high a grade the parenchymatous changes in the kidneys may attain is shown by a case recorded by Siebert:

A man who jumped from the steps of a railroad car when in motion was turned over and over several times and thrown to a considerable distance, where he was found lying on his back. The urine was subsequently scanty, bloody, and albuminous. After six weeks death ensued. The substance of both kidneys was reduced to a pulpy, bloody mass, which could not retain a cut surface, and from which the detritus into which the greater part of both kidneys had been transformed could be squeezed out.

When the disease has existed for a long time, a purulent liquefaction of larger or smaller portions of the kidney takes place. This may involve the whole or a part of one or both kidneys. Such renal abscesses may attain a considerable size, and cases have been reported in which nothing of the kidney was left but the capsule and the calyces, which offer a greater resistance to the process of purulent liquefaction. In these cases it is always exceedingly difficult to determine from the lesions whether the suppurative process began in the kidney itself, or in its pelvis, as a pyelitis, with subsequent extension to the renal substance. Such cases of pyelonephritis have been very often mistaken for renal abscesses. The anatomical discrimination is only possible where a rupture into the renal pelvis has not taken place, or where, notwithstanding this, the pelvis has remained healthy. Only then can we declare with certainty that the renal suppuration was primary. Clinically, the opening of the abscess into the pelvis of the kidney will be revealed by the passage of pus per urethram, and the diagnosis will be more certain if the discharge of pus be sudden and profuse, and the urine had previously contained no pus. The large pus-sacs of the kidney—described by the earlier writers as renal abscesses, and of which Voigtel made a complete collection—should for the most part be classed under the head of purulent pyelonephritis. In addition to the abscesses of the kidney itself, which possess smooth or ragged walls, the anatomical examination reveals a series of lesions which are consecutive to the primary disease. Among these are phlegmonous inflammation of the perinephritic and retroperitoneal connective tissue, which extends irregularly in different directions; burrowing of pus and fistulous abscesses; and finally, adhesions, which form between the kidney and the neighboring organs and ultimately lead to communications, particularly with the large intestine. I have described these lesions more in detail in the chapter on Nephrolithiasis, because they are most frequently met with in connection with that affection. I will here only allude to an extremely rare case observed by Rayer, in which an abscess of the right kidney opened into the liver. Sometimes the contents of renal abscesses, instead of being discharged become inspissated, the fluid portions are reabsorbed, and a sort of cheesy mass is left, which contains shrivelled pus-corpuscles and deposits of the phosphate and carbonate of lime, often in considerable quantities. An abscess can in this manner be rendered permanently harmless, and may continue to exist without further manifestations. The loss of function of one portion of the renal substance is often equalized by a compensatory hypertrophy of the other kidney. In isolated cases the formation of an abscess in one kidney is accompanied by amyloid degeneration of the other. Even simple contraction of the tissues may, as it appears, lead to the absorption and cure of abscesses.

Those abscesses of the kidney whose etiology is still uncertain present the same anatomical appearances as the traumatic abscesses. *Pyæmic* renal abscesses are at all events very rare. In the cases of pyæmia dissected by Waldeyer,[1] no pyæmic abscesses of the kidneys are mentioned. In earlier times pyelonephritis, caused by retention of urine and its resulting conditions, was thought to be a pyæmic abscess, and Beckmann still maintains this opinion. I will return to this point in speaking of pyelonephritis, and now refer the reader to the description there given, because it corresponds completely with the description usually given of pyæmic abscess. I will, however, admit here that, like Cohnheim,[2] I believe that in the course of the disease called pyæmia the kidneys, as well as other organs, may become the seat of very large pyæmic abscesses.

The anatomical appearances presented by the embolic abscesses are rather confused. Generally both kidneys are affected, the organs are enlarged; the capsule, in which, as well as in the cortex, extravasations of blood are often found, is in some parts easily separated, but is firmly adherent at those points where the abscesses now to be described exist. The latter may often be seen through the capsule. The parenchyma is sometimes relaxed, but is generally firmly elastic. At several points, particularly in the cortex, isolated yellowish patches are frequently seen. In general the parenchyma of the kidney is cloudy and grayish red, the cortical substance being especially affected. The characteristic sign of this process, however, is the presence of *punctiform yellowish collections of pus*, *miliary abscesses*, which are mostly surrounded by hyperæmic halos. They are generally raised slightly above the surface, and are often present in enormous numbers, so that the surface appears to be sown with them. Sections through the organ show them to be nearly as numerous in the cortical as in the medullary substance. In the former they are rounded or wedge-shaped, while in the pyramids they are often very much drawn out, or linear. Aside from those rare cases in which infected thrombi of the pulmonary veins are broken loose and carried into different parts of the general arterial system, these true metastatic abscesses are met with almost exclusively in connection with malignant endocarditis sinistra, aortica, mitralis, or parietalis, which produces specifically poisonous emboli. It is characteristic of these remarkable affections, that under their influence the tissue of the valves very rapidly loses its consistency, and becomes so soft and brittle that the force of the blood-current shatters, and, so to speak, grinds it into fine particles which are large enough to block up arteries of the smallest calibre, or perhaps only the capillaries themselves. The abscesses of the kidney are never so large as to require for their production embolism of the larger arterial branches. The pathogenesis of the process is simply this: as a result of a specifically infecting embolism, a true circumscribed inflammation is developed whose product consists of healthy pus. With regard to the source of the pus it appears to me that the facts recorded by Cohnheim compel us to seek it in the migration of the white blood-corpuscles, and, indeed, the vascular theory of inflammation must now be considered the only tenable one. Formerly the proliferation of the connective tissue corpuscles in the kidney, as well as elsewhere, was generally regarded as the source of the pus. Johnson alone maintained that the pus was formed from epithelium, or, as he more guardedly expressed himself, the normal epithelium is replaced by pus. This view has recently found an advocate in Lipsky,[1] his experiments leading him to the conclusion that the epithelium of the uriniferous tubules is the only possible source of the cell production, and that this cell or pus formation can take place in two ways, viz., by division and by endogenous cell-formation. Only in a few instances, says Lipsky, could the possibility of another mode of origin be admitted. In the interstitial tissue, to which this author gave little attention, he took notice only of the excessively distended and evidently dilated vessels between the uriniferous tubules.

---

[1] Zur pathol. Anatomie der Wundkrankheiten. Virchow's Archiv. 40.

[2] L. c. pag. 99.

Recently in a series of these cases, low organisms, which are commonly designated as *bacteria*, have been found in the kidney. They have been found sometimes undoubtedly in the vessels, both in the tufts of the glomeruli and in the distended veins, but also in the midst of the dense masses of white blood-corpuscles within the described abscesses. They are mostly the sphero-, but partly also the rod bacteria. These are introduced into the blood apparently from without, through wounds or by way of the mucous membranes, as for example, from the intestines in cases of mycosis intestinalis, and fasten themselves perhaps upon the valves of the heart, whence they are carried by the blood into the various organs of the body, and among others the kidneys. The question whether these bacteria are to be regarded as the cause of the abscess is of much interest. A large number of observers, and particularly Billroth, regard the development of bacteria as a secondary process, entirely dependent upon the septic inflammatory processes or their products. Other observers have pointed out that a number of cases of endocarditis in which the organisms under consideration were not found, ran the same malignant course. To this it may be objected that it is quite incomprehensible why these small organisms are not constantly discovered in these abscesses, if they are always the product of septic inflammatory processes. It will be, moreover, readily admitted that infecting substances of different kinds, parasitic and non-parasitic, may produce the same results.

---

[1] Wiener med. Jahrb. 1872. 2. Heft. S. 155.

I do not, however, *si duo faciunt idem, non est idem*, think that the facts we at present know, and the *à priori* reasoning, are sufficient to enable us to form a decided opinion on this difficult question. I will only mention that C. Weigert[1] has shown that sometimes in small-pox, in the neighborhood of the collections of bacteria found in the lymph-glands, the spleen, the liver, and the kidneys—the bacteria in the two last being seated in the blood-vessels—the nuclei of the cells have disappeared and the cells themselves are often destroyed. Although there is the greatest difference between this condition and an abscess, it affords evidence nevertheless that these organisms are not always as innocent and harmless as many authorities believe. It cannot as yet be determined whether they are only the carriers of, or themselves constitute, the poison. At all events I consider it advisable to remain non-commital on these questions, which are still only in the commencement of their development.

---

[1] **Tageblatt d. 47. deutsch. Naturforscherversammlung. Breslau. 1874. S. 277.**

Symptomatology.

I will first describe the symptoms of traumatic purulent nephritis. These vary greatly, according to the intensity of the injury. Celsus[1] himself made an attempt to collate the manifestations which are diagnostic of injury of the kidney: *Renibus vero percussis dolor ad inguina testiculosque descendit, difficulter urina redditur, eaque aut est cruenta aut cruor fertur.* The cases have been divided, according to their duration, into *acute* and *chronic*. This classification is not bad, for there are many points of difference between cases that run a rapid and those that run a slow course. Frequently the several symptoms follow one another with extraordinary rapidity. The beginning of suppuration is generally marked by a chill. The fever is a continuous one, and is often interrupted by chills. The digestion suffers from the very start, there is a bad taste in the mouth, the appetite is lost, and not unfrequently there is repeated vomiting of a slimy and bilious material. According as one or both kidneys are affected by the injury, a severe pain is soon experienced in one or both loins. The pains vary very greatly in different cases. Sometimes they are excited by pressure, especially when made in the lumbar region, but often also by deep pressure on the abdomen. Every jarring of the body aggravates the pains, and often even the respiratory movements and the pressure of the abdominal walls in defecation have the same effect. Lying upon the affected side, when the affection is unilateral, increases the pain, as in pleurisy. When both kidneys are affected, the lateral and the supine positions are alike painful. The pain is permanent, more or less piercing, or dull. It is frequently radiating, sometimes upwards towards the diaphragm, the colon, the liver, the spleen, even to the breast and the shoulders, but most frequently downwards, following the course of the ureters, into the bladder, or into the thigh of the affected side, when it is accompanied by a feeling of numbness and stiffness. In male patients, even when there is no nephro-lithiasis, a retraction of the testicle of the affected side toward the inguinal ring is observed, in consequence of spasmodic action of the cremaster, whilst in females the pain often radiates towards the insertion of the ligg. rotunda.

---

[1] Lib. V. Cap. 26. Nr. 11.

In addition to the pains, *the changes in the urine* are of especial moment. Very often it contains blood for a longer or shorter period after the injury. It is at the same time extremely scanty, and, if both kidneys are affected, may be entirely suppressed. These are the worst cases, and are almost invariably fatal. A so-called adynamic fever, a status typhosus, is developed, which proves rapidly fatal, with coma and other symptoms of uræmia. In other cases pus is passed pretty early with the urine, blood and pus being frequently passed at the same time. The chemical changes of the urine in purulent traumatic nephritis have not yet been studied with sufficient minuteness. It is feebly acid, or, in rare cases, neutral or alkaline, according as it contains pus and blood or the products of commencing decomposition. A diminution of the urates and uric acid has been observed.

In other cases the course of the traumatic renal inflammations is much *slower* and *more tedious*. The acute manifestations, pain, etc., disappear, but the passage of bloody urine may still continue for months, or the blood may be replaced by pus. In this way a high degree of marasmus is often produced, and the patient finally dies, exhausted by the long-continued discharge.

However, the traumatic renal inflammations do not always terminate so unfavorably. Troja reports cases in which portions of the renal substance protruded through the wound, and nevertheless the patients recovered. Treyden saw a case of punctured wound of the kidney, in which bloody and purulent urine was passed, which recovered.[1] Meoli treated a young girl who was wounded in the right kidney with a cutting instrument. Bloody urine was passed, and on the twelfth day high fever set in, which continued until the twenty-first day. The wound suppurated very profusely, but gradually contracted, until it became a mere fistulous opening, and finally closed.[1] Rayer also reports some cases of recovery from traumatic nephritis—not only cases in which the inflammation was excited by blows and kicks, but also the case of a boy, eleven years of age, in which inflammation and suppuration in the kidney followed a wound of that organ produced by a pocket-knife. Although suppurative fever, emaciation, and diarrhœa had brought the child nearly to death's door, recovery took place in about two and one-half months. Recently Schuster reported an analogous case:[2] a butcher's apprentice received a stab in the lumbar region, which caused hematuria and urinous discharge from the wound, with corresponding diminution of the renal secretion; but the wound healed gradually within two months. It is unnecessary to multiply these instances.

---

[1] Rust's Magazin. Bd. XVII. S. 1.

The wounds of the kidney, with consecutive suppurative nephritis, which end in recovery, are generally those which have been inflicted from behind. The injury in these cases is more likely to be confined to the kidney, while wounds inflicted from the front are necessarily complicated, and almost invariably fatal. Gunshot wounds of the kidney and the resulting traumatic nephritis are under all circumstances very dangerous, albeit even here cases of recovery are not lacking. Pendleton[3] relates a case of gunshot wound of the kidney, where the bullet entered the body about two and one-quarter inches to the right of and below the navel. No aperture of exit could be found. The diagnosis was based upon the passage of bloody urine. Later colicky pains set in in the region of the kidney. Under the use of opiates, recovery ensued.

In those cases in which renal abscesses are developed without any satisfactory cause being discernible, and which have been attributed more frequently than appears warrantable to rheumatic influences, the symptoms are developed slowly, and the clinical picture is often anything but clear. Febrile attacks, vomiting, and pain in the loins may exist for months, and even years, during which time the patient may be confined to his bed only at intervals, or perhaps not at all. The digestive disturbances are very apt to become prominent, especially at the time of the usually severe febrile attacks. Fever is a constant accompaniment of large renal abscesses, but if the suppuration be of limited extent, it may run its course either entirely without fever, or, at most, be accompanied by very slight elevation of temperature. Sometimes the fever is the only objective symptom of the renal abscess. Vogel relates the history of a man, aged thirty-six years, who was seized with the symptoms of a gastro-rheumatic fever. His condition rapidly improved, but suddenly a pretty abundant purulent sediment was observed in the urine. This continued for weeks, without being accompanied by the least difficulty in micturition. Later, pain in the region of the kidney and frequent rigors set in. An intercurrent typhus carried off the patient. The section showed an almost complete destruction by suppuration of the parenchyma of one kidney, without any other anomaly in the uropoëtic system. Pus, however, is by no means always present in the urine when purulent collections exist in the kidneys.

---

[1] See *Naumann*, l. c. p. 33.

[2] Oesterr. Zeitschrift. f. prakt. Heilk. 1868. Nr. 12.

[3] New Orleans Journ. of Medic. Oct. 1867.

In addition to the symptoms so far enumerated, a *tumor of the diseased organ*—a pus-sac—is developed in the course of a purulent nephritis, which may be perceptible to external examination. This usually occurs only when the suppuration has existed for a long time. The size of the tumor will be in proportion to the amount of renal parenchyma which is involved in the inflammatory and suppurative processes, and the quantity of pus that has been discharged externally. The pus will be discharged externally through the urethra, when a communication takes place between the abscess and the renal pelvis, provided the urinary passages are patent. If the latter, from any cause, be temporarily obstructed, the discharge of pus will cease. With this alternating appearance and disappearance of pus in the urine, various local and general manifestations are connected:

pains and a feeling of tension in the tightly distended tumor, chills and digestive disturbances, which cease as soon as a free outlet for the pus is secured. The discharge of purulent urine often excites frequent desire to micturate, and pain in the end of the penis. The escape of the pus from the abscess into the pelvis of the kidney takes place suddenly, and generally a large quantity escapes at once. Under favorable circumstances, after the abscess has been emptied, its walls may contract and cease to pour out pus, and cicatrization ensue. At all events, the discharge into the renal pelvis is more favorable for cure than is perforation into other organs. Todd recounts in his clinical lectures, page 394, a case of this sort, which left the hospital at least much improved. The constitutional symptoms were, in this case, but slightly marked, and Todd classes it among the milder cases.

The patient was a married woman, twenty-nine years of age, who two years previously was attacked with severe pains in the left side, loss of appetite, and fever. Later she noticed a tumor in this side, and afterwards found blood and pus in her urine. The tumor gradually enlarged and became painful. Suddenly one day she experienced a sensation as if the tumor had burst, and it emptied itself through the urethra. After a fortnight's sojourn in the hospital, with the help of rest, good diet, and small doses of quinine, her condition had decidedly improved.

It might not be amiss to draw attention to the fact that a sudden discharge of pus in considerable quantities through the bladder is not altogether diagnostic of renal abscess. A single example will suffice to recall to the reader's mind one of the numerous ways in which this may be brought about. The case is reported by Ogle.[1] During life the existence of a renal abscess of the right side seemed to be indicated by pain in the region of the kidney, vomiting, chills, and purulent urine. The post-mortem revealed a large abscess behind the left kidney, starting from the carious second lumbar vertebra; the pus had gravitated down to the left side of the bladder, and made its way into that organ through several small round openings. A circumscribed collection of pus was also actually present in the right kidney.

---

[1] St. George's Hosp. Reports, 1867, p. 371.

Not one symptom indicating disease of the vertebral column had existed during life.

In these cases of suppurative inflammation of the kidneys, small portions of the renal substance are sometimes separated, dissected out as it were by the process of suppuration, and are discharged with the pus through the urethra. The ancients believed that these necrotic pieces of the renal parenchyma were the carunculæ renales. This is not a very frequent occurrence.

Taylor[1] relates the history of a boy who, after passing through an attack of scarlet fever one and a half years previously, fell into a state of chronic marasmus, and complained of pains, especially in the left half of the abdomen, along the course of the ureter. The scanty but frequently voided urine often contained pus, but never blood. On one occasion a rounded, soft, grayish, ragged body, which was partially decomposed and weighed over five drachms, was passed per urethram with much straining, and as its microscopical examination showed Malpighian capsules and a few uriniferous tubules with distinct epithelium, it unmistakably consisted of kidney substance. The patient died eleven weeks later. The section revealed a perforation between the right kidney and the ascending colon, and dilatation of the ureters, renal pelves, and calyces upon both sides. The renal substance was soft, and portions of it were broken down into a purulent mass, while here and there distinct loose pieces of the parenchyma, which closely resembled in appearance the body previously passed with the urine, were almost completely separated by the process of suppuration.

Wiederhold, of Cassel, also publishes a somewhat similar case.[2]

A patient, in whose urine pus and albumen had existed in abundant quantities for some time, one day discharged a cloudy urine which deposited a heavy sediment, in which a ribbon-like coil of tissue the size of a pigeon's egg was found. The microscopic examination showed this to consist of renal substance, in which the uriniferous tubules could still be very distinctly demonstrated. The patient was suffering from an abscess in the neighborhood of the left kidney, from which the suppurative process had extended to the kidney itself. The patient lived two years after the passage of the piece of kidney.

Concerning the perforations of renal abscesses in other directions, I may in general, in order to avoid repetition, refer to what I shall say, under the head of Nephrolithiasis, about the abscesses caused by renal concretions. The traumatic renal abscesses, and also those whose etiology is uncertain, may rupture into the retroperitoneal connective tissue, and there give rise to widespread phlegmon and deep burrowing of pus. They may—but, fortunately, this is rare—rupture into the peritoneal sac and cause a rapidly fatal peritonitis diffusa. After the perforations into the renal pelvis, those into the colon are the most frequent.

---

[1] Ref. in Schmidt's Jahrb. 114. Nr. 4.

[2] Virchow's Archiv. Bd. XXXIII. S. 552.

Portal reports the case of a man, aged sixty, in whom a perforation and communication existed between the kidney and the colon. In a case reported by Gintrac[1] a similar condition existed.

The patient was a woman, forty-eight years old, and mother of nine children. Two years before her death febrile attacks, vomiting, and diarrhœa, alternating with constipation, set in without assignable cause. At the same time she complained of pain in the loins and in the abdomen, especially on the left side. The symptoms recurred at long intervals. One year before death the attacks became more frequent and severe. A tumor of the left kidney was discovered. The patient became emaciated and very weak. Eight days before death she had several large fluid passages, and the volume of the tumor suddenly diminished. The fever, increasing weakness, and emaciation, soon caused death. The left kidney contained in its lower part an abscess which communicated with the colon through an opening one and a quarter inches in diameter.

Perforations of renal abscesses into the respiratory organs are extremely rare.

Heer,[2] however, has described an instance of this which occurred in Meckel's practice. The patient lived ten years without great suffering. The disease had developed after a fall upon the left side of the body. The patient at last began to expectorate purulent material, and died of phthisis. The left kidney presented a sac, the size of a child's head, which was filled with pus and was firmly adherent to the diaphragm, and also to the left lung. The latter contained an abscess which communicated with the renal abscess.

Rayer reports two analogous observations. Lennepveu[3] has also collected several cases of perforation of renal abscesses into the bronchi.

As a result of renal abscesses, especially those which run a chronic course and are complicated with disease of the bladder and the urinary passages, *paralyses* are occasionally developed which in general have the pronounced characters of spinal paralysis and hasten the fatal termination. The term *paraplegia urinaria*, or *urogenitalis*, which explains nothing, is usually applied to them. This symptom did not escape the observation of the earlier writers. Thus Troja says:

---

[1] Union médicale. 1867. Nr. 48.

[2] De renum morbis. 1790. p. 27.

[3] Sur les fistules réno-pulmonaires. Thèse. Paris. 1840.

"During the course of a severe renal inflammation the irritation of the nerves of the kidney may be propagated to the spinal cord, and in this way paralyses of the lower extremities, with loss of sensibility and movement, which ended in death, have been occasionally produced."

It is particularly interesting to us to know that Troja laid stress upon the affection of the spinal cord, which he observed in one of these cases. Stanley, however, first called the attention of the profession to these forms of paralyses. They had long been classed among the so-called reflex paralyses which arise without appreciable disease of the spinal cord, in consequence of some peripheral affection. In reality the greater part of them are dependent upon affections of the nerves and the spinal cord. More detailed descriptions of these interesting forms of paralyses belong to the department of spinal and nervous diseases. The paralysis that is secondary to suppurative nephritis usually takes the form of paraplegia, but paralysis of one leg is also observed in isolated cases. We find, *e. g.*, a case of this sort reported by Siebert.[1]

A man, aged thirty, suffered from paralysis of the right leg, with dull, and at times shooting pains in it. No disturbance of nutrition or change of temperature could be detected in the leg. The patient also had frequent chills, high fever, sweating, and dry râles over the lungs. The suspicion that it was a reflex paralysis, and that its cause was to be sought in an affection of the kidney, was strengthened by the presence in the urine of a greenish purulent sediment an inch in depth. The patient stated that he had formerly suffered from renal colic, which ceased two years before, when the paralysis of the leg began. The autopsy revealed an immense abscess of the right kidney; the capsule was filled with thick pus, and no trace of the renal substance remained. The left kidney was larger than normal, but perfectly healthy. Unfortunately, no mention is made in this report of an examination of the nerve plexus or of the spinal cord.

---

[1] L. c. p. 382.

The small *metastatic abscesses*, which are produced by embolism of the renal vessels with infecting substances, and also the so-called pyæmic abscesses, which are not of embolic origin, do not give rise to any characteristic symptoms. The albumen and casts which are occasionally found in the urine in these cases are not dependent upon the circumscribed collection of pus in the kidney. The symptoms, which would indicate with some probability the existence of renal abscesses in connection with these diseases, are marked pain and tenderness in the region of the kidney, and the discharge of considerable quantities of pus with the urine.

Complications.

Traumatic nephritis is often complicated with other grave injuries, which may easily distract the attention from the kidneys.

Chronic renal abscess of one side, like suppurative processes elsewhere, sometimes excites amyloid degeneration of the other kidney.

Diagnosis.

The diagnosis of traumatic nephritis rests upon the history of injury, and the passage at first of blood, and afterwards of purulent urine. To these may be added the exceedingly great local tenderness and the other signs mentioned under the head of symptomatology.

The diagnosis of idiopathic renal abscess, for which no adequate cause can be discovered, is exceedingly difficult, and frequently even impracticable, not only in the earlier stages, but also throughout its entire course. Fever, occasional chills, gastric disturbances, vomiting, loss of appetite, and diarrhœa, are often the only objective symptoms. It is only when in addition to these symptoms there is tenderness over the region of the kidney, and a tumor can be made out which is evidently connected with that organ, or when rupture of the abscess occurs, that a diagnosis becomes possible. Even under these circumstances experience has taught us that there are many sources of error, which cannot be eliminated by the most careful consideration of the symptoms and etiology. As an illustration of the very great difficulty, and in fact occasional impossibility, of making a correct diagnosis, I may refer to the case reported by Ogle, which will be found on page 556.

That the renal suppuration occurring in connection with the disease called pyæmia, and also the embolic metastatic abscesses, cannot be diagnosticated, but only surmised, has already been stated.

Duration, Termination, and Prognosis.

The duration of traumatic nephritis suppurativa is very variable. Some cases, and especially those which are complicated with other severe injuries, terminate fatally in a very short time. On the other hand, we meet with a series of cases in which the acute stage with its attendant dangers is happily tided over, and the disease becomes chronic. This is sometimes the case with the traumatic, but more frequently with the idiopathic renal abscesses, many of which last for years. That the embolic and pyæmic abscesses should always run very acute courses, necessarily follows from the nature of the primary diseases. The terminations of suppurative nephritis are usually bad. When the abscess is of traumatic origin, many patients die during the first few weeks. Others succumb after the disease has become chronic, in consequence of exhaustion. The cases in which permanent cures occur are rare, but seem to be most frequently of the traumatic variety. The prognosis is therefore from the start more than dubious, and in most cases bad.

Treatment.

The treatment is most likely to be successful in cases of acute traumatic nephritis. Here, if the patient can bear it, a strongly antiphlogistic treatment may be tried. In every case absolute rest, ice applications, and unirritating diet are indicated. In this way we may sometimes succeed in preventing the widespread suppuration of the renal substance, or at least in confining it within moderate bounds. In all other cases the treatment must be purely symptomatic. It can hardly be necessary to add that treatment is useless when the abscesses are of pyæmic or metastatic origin.

Pyelitis and Pyelonephritis.

History and Literature.

Rayer first separated the inflammations of the pelvis of the kidney from those of the renal substance. I describe pyelitis and that form of nephritis which is produced by extension of inflammation from the renal pelvis to the renal parenchyma, in the same chapter, because this extension of inflammation very frequently occurs, and a separation of the two conditions is often practically impossible. The proofs of this will be found farther on.

In addition to the authors mentioned on page 543, I have consulted in preparing this chapter: *Henoch*, Klinische Wahrnehmungen. Berlin. 1851. S. 209.—*Todd*, Clin. lect. on diseas. of urin. organs. 1852.—*Oppolzer*, Wien. med. Wochenschrift. 1860.—*Mosler*, Archiv der Heilkde. 1863. S. 420.—*Treitz*, Prager Vierteljahrschrift. 1859.—*Jacksch*, Prager Vierteljahrschrift. 1860.—*Traube*, Beiträge zur Pathol. u. Physiol. II. S. 654.—*The same*, Symptome der Krankh. der Respirat. u. Circ. Apparats. S. 117.—*The same*, Berl. klin. Wochenschrift. 1874. Nr. 4.—*Ferber*, Virchow's Archiv. 52.—*Fürstner*, Virchow's Archiv. 59.—*Kaltenbach*, Archiv f. Gynäkol. III.—*Stadfelt*, Schmidt's Jahrbb. 157. S. 57.—*Malherbe*, De la fièvre dans les maladies des voies urinaires. Paris. 1873.—*Ollivier*, Arch. de physiol. V. p. 43. 1873.—*Dickinson*, Med. chir. transact. LVI. p. 223-234. Schmidt's Jahrbb. 161. S. 264.—*Leyden*, Rückenmarkskrankheiten, I. 1874.

Etiology.

The etiology of pyelitis and pyelonephritis is of very great practical importance, because the symptoms and the course of these affections, the conditions which render the course acute or chronic, favorable or unfavorable, are closely related to and directly dependent upon the exciting causes of the inflammation. The individual cases have often very little clinical affinity with one another. At times the symptoms of the pyelitis are prominent, but often it runs an entirely or partially latent course, as a local manifestation of some severe constitutional affection. As instances of the latter, I may mention the cases of pyelitis which are developed more or less constantly as complications of typhus, typhoid, the exanthemata, the so-called pyæmic complex of symptoms, diphtheria, cholera, carbuncle, and scurvy. Further, pyelitis not infrequently occurs in connection with acute or chronic morbus Brightii. In diabetes mellitus, also, we occasionally meet with a secondary inflammation of the renal pelves. Pyelitis, as well as irritation of the renal parenchyma and of the urinary passages, may be excited, primarily, by the use of immoderate doses of balsam of copaiba, cubebs, oil of turpentine, and other severe diuretics, especially cantharides. I mention this here only incidentally, because the organ on which the injurious action of these drugs is exerted with the greatest intensity is the bladder (compare Diseases of Bladder). The irritation is mostly very temporary, and its clinical importance is slight.

Far more important are those cases of pyelitis which are excited by the *irritation of foreign bodies in the renal pelvis or the renal calyces*, and which, by extension, often involve the renal parenchyma. At the head of this class of causes are the renal calculi, which excite not only the most severe, but also the most frequently observed forms of pyelonephritis. Far rarer are the cases of pyelitis, and eventually, pyelonephritis, which are caused by animal parasites (with us usually the echinococci), by retained blood-clots, and by malignant growths of cancerous or tubercular nature. I have, for the sake of expediency, in order not to destroy the unity of the clinical pictures, more fully considered all these forms under the heads of Nephrolithiasis, Animal Parasites of the Kidney, Renal Cancer, etc.

Those forms of pyelitis and pyelonephritis which arise when from any cause *ammoniacal decomposition of the urine* takes place, are of great clinical and practical importance. In the majority of cases this decomposition is a result of retention of urine. Whatever hinders the regular discharge of the urine predisposes to retention. I will mention here a few of the numerous conditions that produce this effect. One of the rarest is congenital phymosis. Interesting in this connection is the history recounted by Mosler of a man aged eighteen years. The entire right kidney formed an ulcerating cavity, with irregular walls, the medullary substance being entirely and the cortical substance partially destroyed. The left kidney was entirely absent. Death was due to uræmia. Far more frequently, obstruction to the discharge of urine, and its consequent ammoniacal decomposition, are due to strictures in the posterior part of the urethra, enlargements of the prostate, etc. Retention of the urine in the bladder is often also due to paralysis of the organ, in consequence of spinal disease. Decomposition of the urine often sets in very rapidly in these cases. On the other hand, severe inflammations of the bladder—*e. g.*, those caused by calculi—not infrequently cause decomposition of the urine, even without retention. Decomposition of the urine from this cause always leads to the formation of phosphatic sediments, consisting especially of the well-known crystals of the ammonio-magnesian phosphates, and then a vesical catarrh is excited. This catarrh of the bladder keeps the urine constantly alkaline, and a rapid, continuous decomposition of the urine, which becomes excessively fetid, sets in, accompanied by an active development of vibriones and bacteria. Recently a rôle of great importance has been attributed to these bacteria in the causation of pyelonephritis and pyelitis. I will defer the consideration of this point until I have described the pathologico-anatomical lesions.

Pyelitis and pyelonephritis in the female sex require special consideration, on account of their connection with *affections of the sexual organs.* They are developed after obstetrical operations, in the puerperal stage, and during pregnancy. I may state in advance that a large number of these cases are really due to retention of urine. During pregnancy a catarrh of the bladder may extend to the ureters and pelves of the kidneys. The primary catarrh may be caused by the irritation produced by the pressure of the fœtal head on the neck of the bladder, in the narrow pelvic inlet, or by the retention consequent on retroversion of the gravid uterus. Perfectly idiopathic catarrhs of the urinary passages, which may extend to the renal pelves, are sometimes developed, with marked fever, during pregnancy, under, as yet, uncertain etiological conditions.

General catarrhal affections of the urinary passages, with the symptoms of pyelitis, may occur as apparently perfectly independent *idiopathic* affections during the puerperal period. They do not appear to be very rare. Their pathogenesis is still obscure; perhaps they are connected in some way with the processes of involution that take place in childbed. On the other hand, pyelitis in the puerperal state may depend on extension of *inflammation from neighboring parts*, as, *e. g.*, from a perinephritis, which now and then complicates puerperal parametritis. Sometimes the inflammations of the renal pelvis are mere extensions of simple vesical catarrhs, which spread upward in the direction of the urinary passages. Finally, in cases of parametritis, or of inflammatory swelling of the connective tissue that binds the uterus to the bladder, pyelitis is often caused mechanically by compression of the ureters, either at their openings into the bladder, or before they reach that organ, by the inflammatory exudation, and consequent retention of urine. Further, the openings of the ureters become displaced and compressed by the great swelling after operations upon the cervix or the anterior vaginal wall, in which the adjacent part of the bladder, the region of the trigonum, is involved. A temporary retention of urine is produced, and, later on, an extension of the catarrh to the renal pelves.

In a few cases catching cold may be looked on as the cause of pyelitis. Rosenstein regards the moist climate of Groningen (Holland) as probably a very important factor in the etiology of the pyelitis which so frequently occurs there.

Pyelitis and pyelonephritis occur at every period of life. It is rarer in the young than in adults, and is oftener observed in men than in women. This naturally follows from the fact that renal calculi, and especially retentions of urine, the two principal exciting causes of inflammation of the renal pelvis, are more frequently met with in men.

Latterly Ollivier has described a "new" variety of pyelonephritis, which is excited by blood coagula, that produce the same irritation as foreign bodies. He classes it with the pyelonephritis caused by foreign bodies, which in these cases consist of fibrinous concretions. It is said to be especially met with in old persons. The cause of the bleeding is to be found in atheroma of the arteries. In advanced age atheroma of the arteries is said often to occasion renal hemorrhages (comp. Diseases of the Renal Arteries).

Pathology.

Anatomical Changes.

I shall not consider in this place the pyelitis and pyelonephritis induced by the mechanical irritation of large solid bodies, *i. e.*, of renal concrements, animal parasites, etc. These will be considered in their appropriate places.

In those forms of pyelitis which occur primarily or secondarily as complications of infectious diseases, etc., we usually find the signs that characterize the catarrhal inflammations of other mucous membranes. The blood-vessels of the mucosa are greatly distended, and more or less dilated, and the surface of the mucous membrane is covered with mucus, or a muco-purulent secretion. Again, *diphtheritic forms* of pyelitis are sometimes met with in connection with severe constitutional diseases, particularly with puerperal diseases. Extensive yellow spots, which consist of mortified tissue, are then seen upon the mucous membrane of the renal pelvis. This destructive process usually extends for some distance into the papillæ of the medullary substance.

Those forms of pyelitis and pyelonephritis which are caused by *retention of the urine* and its *ammoniacal decomposition* are preceded by an affection of the bladder—a vesical catarrh which may, however, be cured during life, so that no signs of it may be visible at the autopsy. Both ureters may be implicated, but sometimes they also are healthy. The appearances in the renal pelvis in the earliest stages are the same as those described above—hyperæmia and increased secretion of mucus and pus. In the severer forms extravasations of blood are found in the tissue of the mucous membrane. In these cases the inflammation is rarely limited to the renal pelves, the kidneys being generally more or less involved. In *recent* cases the kidneys—

sometimes one, but often both—are slightly enlarged. The capsule, as a rule, can be torn off with ease and without loss of substance. The organ is distended with blood, and its surface is very red, but at the same time presents more or less numerous pale spots from one to three centimetres in diameter. Upon section of the organ, narrow, white, wedge-shaped portions of tissue are seen, which can be traced through the thickness of the cortical into the medullary substance, and grow smaller towards the apex of the medullary cone. The changes of the kidney in this stage rarely come under the observation of the pathological anatomists, since the process, as a rule, only proves fatal in the more advanced stages. In these later stages the pale portions just described are more swollen, and the fibrous capsule is more difficult to separate at the points corresponding to them. At the same time yellow, punctiform, purulent foci present themselves in the swollen portions, which upon section of the organ are seen to correspond to small abscesses that extend in the direction of the uriniferous tubules. Through the confluence of several of these small abscesses larger pus cavities are formed. In other cases the purulent softening of the renal tissues begins in the pyramids. In this manner large abscesses are formed, which constantly increase in size, and involve also the cortical substance, so that finally, instead of the kidney, only a thick-walled sac remains, which is filled with pus, and incompletely partitioned by septa which correspond to the several renal calyces. Hand in hand with these destructive processes, cicatrization and contraction take place to a greater or lesser extent. Fibrous streaks and bands may be developed, which, in spite of their occasional wedge-shaped form, can easily be distinguished from an embolic lesion, because they do not correspond in extent to the limits of a vascular territory, and the apex of the cicatrix usually extends into the pyramids. Sometimes, in connection with the cicatricial processes, there is a diffused increase of the interstitial connective tissue with simultaneous transformation of the capsule into a tough, callous, greatly thickened mass.

Upon microscopic examination of the kidneys in the earlier stages of the process we can see, even with low magnifying powers, dark lines with the characteristic contours of the convoluted tubules. With higher powers the uriniferous tubules are seen to be filled with small shining granules. These granules are often arranged very regularly in parallel lines, or in lines radiating from a central point. They are brilliant, highly refractive, and are not affected by the addition of acids, alcohol, alkalies, or ether. The uriniferous tubules appear wider than usual, and the epithelial cells are somewhat enlarged, very cloudy, and perhaps in a state of fatty degeneration. At certain points, where granules spread out in divergent lines from the epithelium, the cells appear in every diameter smaller than normal, and, as Klebs believes, increased in number by a process of segmentation. All interstitial changes are absent in this stage of development of the process. In the later stages microscopic examination shows that the interstices between the individual uriniferous tubules are also widened, and often filled with the same shining granules as those observed in the tubules, and, besides these, with more or less numerous pus-corpuscles. The epithelial cells of the uriniferous tubules are now destroyed, and here and there branching filaments of fungi are observed, besides the spores. Pus-cells are often present within the medullary substance, especially in the vicinity of the papillæ; and at these points they must be derived chiefly from the renal pelvis, since the looped tubules contain fibrinous cylinders without pus-cells. The larger yellow foci present the appearance of ordinary abscesses under the microscope. They consist of closely packed pus-corpuscles lying alongside of one another, partly well preserved and partly broken down. Often, however, the remains of sharply outlined uriniferous tubules, filled with the smallest granules, are seen, sometimes in the centre, sometimes at the periphery of the abscess. In the vicinity of the abscess we also often find collections of altered blood-corpuscles, which, like the pigment, point to previous hemorrhages.

I can entirely corroborate by my own experience this description, which is based upon that first given by Klebs. There can be no doubt that in the small granules here described we have to deal with colonies of so-called *bacteria*. In my opinion, no doubt can remain in the mind even of the most skeptical observer, upon this point, when he sees these low organisms lying in their peculiar grouping within the lumina of the uriniferous tubules, before the destruction of the epithelial cells, which does not occur in the earlier stages, has yet taken place. They cannot, therefore, be confounded with detritus by any possibility. If we inquire now how these bacteria get into the renal pelvis, and what part they have in the development of these pathological processes, we will find these questions answered in the statement of Traube, that the germs of bacteria reach the bladder through infection from without, generally by the introduction of dirty catheters (but also in other ways), and there excite alkaline fermentation of the urine, under the influence of which the urea is changed into the carbonate of ammonia, which in its turn excites inflammation of the mucous membrane. These bacteria reach the pelvis of the kidney through the ureter, and from there wander into the uriniferous tubules. Traube still maintains the view that it is not these microscopic organisms themselves that excite the inflammation, but certain materials which are first liberated by the action of these immigrated parasites upon the substance of our bodies—as an example of which, we may indicate the decomposition of the urea in the bladder. Of the several different views, however, that are still held upon this question, the one defended by Klebs has now the most adherents, and appears to me to be the most probable. He believes that *the bacteria themselves excite the inflammation, causing in the first place a purulent pyelitis, and subsequently circumscribed renal inflammations.* Klebs, therefore, designates this form of renal inflammation pyelonephritis parasitica. The very occurrence of circumscribed foci of inflammation and collections of pus indicates the presence of exciting causes irregularly dispersed through the renal parenchyma; and the development of these foci in the vicinity of the colonies of bacteria, which can be so satisfactorily studied in the earlier stages of the process, proves the necessity of admitting that these minute organisms constitute those exciting causes. It is quite possible that we have to deal with colonies of infected bacteria, which, like infected thrombi, excite phlegmonous inflammation.

The autopsy, moreover, discloses the pathologico-anatomical lesions which cause the retention of urine, viz., catarrh of the bladder, stricture of the urethra, hypertrophy of the prostate, spinal affections, etc. It not unfrequently happens that there is no continuity of disease between the bladder and the pelves of the kidneys—in other words, that the ureters are found to be perfectly free from disease. This circumstance led a number of observers, especially Beckmann, to attribute these secondary affections of the kidneys to a supposed infection of the blood. If we remember, however, as Klebs urges, that the bacteria which excite the inflammation, at least at a certain period of their development, are organisms endowed with the power of independent motion, and are consequently able to spread themselves over wide extents of mucous membrane, until they finally settle permanently in certain places, we will be able to understand how a pyelonephritis may be developed by an exciting cause that is conducted to the renal pelvis from the bladder through the ureter, although the ureter itself remains perfectly healthy, as proven by post-mortem examination.

Symptomatology.

As I will fully discuss the symptoms of calculous pyelitis and pyelonephritis in the chapter on Nephrolithiasis, and of those forms of the disease which are excited by animal parasites in their own appropriate place, it remains for me to describe here in the first place the symptoms of the so-called *catarrhal pyelitis*, which is excited by rheumatic influences; also those of the *secondary* pyelitis, which is met with usually as a sequel of some infectious disease; and finally, and most particularly, the symptoms of those cases of pyelonephritis which are developed in consequence of *retention of urine* (from whatever cause) *combined with its ammoniacal decomposition.* I shall also add a short description of the pyelitis which occurs during pregnancy, or in the puerperal period, or after obstetrical operations, in order to draw the attention of physicians to this interesting complication, which has as yet received very little consideration.

In catarrhal pyelitis the condition of the *urine* constitutes the most prominent symptom. It is always acid as long as the pyelitis remains uncomplicated. In a number of cases the quantity of urine is increased, especially in those cases which are developed after infectious diseases. However, this is not a symptom of the pyelitis. The specific gravity varies according to the quantity of urine. In acute pyelitis *blood* is found in the urine, but never in large quantity. Besides this, the *mucus in the urine is increased* in quantity, and at a very early period it contains pus. The pus is deposited as a whitish, easily recognizable sediment upon the bottom of the glass. That urine which contains blood and pus must at the same time contain *albumen*, requires no explanation. In addition to these, we often find in the urine the flattened, laminated, caudate *epithelial cells of the renal pelves.* Unfortunately, *these characteristic bodies, which are of the greatest diagnostic importance when they are present, are entirely absent in a great number of cases.* The quantity of pus increases slowly and gradually with the advance of the disease. Besides the changes in the urine, *pain* is a symptom of pyelitis, which, however, is often absent, and rarely attains a high grade in uncomplicated cases. It manifests itself as a sense of weight or pain in the region of the kidneys, whence it not unfrequently extends downward toward the bladder. Slight febrile movements and digestive disturbances are also often present. When the catarrhal pyelitis is consequent to some other affection, particularly if this be an infectious disease, its symptoms will be overshadowed by those of the general affection, if the physician neglect the examination of the urine. I have particularly noticed this in abdominal typhus, where I have not infrequently recognized intra vitam slight degrees of pyelitis solely from the urine, and have often verified the diagnosis by the subsequent autopsy. Adequate observations are wanting concerning the symptoms of the *croupous* and *diphtheritic forms* which now and then complicate particularly severe general affections. They are most entirely overshadowed by the other severe symptoms of the primary disease.

Of much greater practical importance is the symptomatology of those cases of pyelitis and pyelonephritis which are developed secondarily after *retention of the urine* and its *ammoniacal decomposition.* Here the affection of the kidney and the renal pelvis is a complication of a primary disease, which in the majority of cases (strictures, hypertrophy of the prostate, catarrh of the bladder, chronic spinal diseases, etc.) has existed for a long time, but which in some rare instances is of recent and acute development, *e. g.*, spinal affections of traumatic origin, and operations on the bladder. It is unnecessary to consider in this connection the symptoms of these different primary diseases. It is evident, however, that in these cases we will no longer find the above-mentioned peculiarities of the urine which are so important for the diagnosis of catarrhal pyelitis. We are no longer able to distinguish the pus-corpuscles and epithelial cells in the ammoniacal urine in which the numerous triple phosphate crystals are embedded in a thick, mucoid, gelatinous sediment. In the microscopic field swarm numberless bacteria. The filtered and acidulated urine contains albumen, as is natural from the admixture of pus serum and eventually of blood. The elementary bodies are destroyed in the ammoniacal menstruum. Two groups of symptoms are developed, however, in this form of pyelitis—at one time *fever*, and, at another, *certain nervous manifestations*. The opinion has now and then been expressed that the fever, which the French call "uræmic fever," always indicates that the kidney is involved; but this is an error. The fever appears in two different forms. One form is acute and attended by great elevations of the temperature, which vary in duration and in their mode of onset, while the other runs a slower course, as a sort of febris continua with remittent character. The facies is anxious, the color of the face yellowish. The nervous manifestations are completely analogous to those observed in the uræmic complex of symptoms. Headache, somnolence, sopor, and delirium exist—in short, the symptoms of typhoid prostration, under which the patients succumb. Frequently there are severe vomiting and diarrhœa, and the tongue becomes fissured and crusted. Pain in the region of the kidneys is rare. Moreover, the disease may excite peri-articular, articular, or diffuse intermuscular inflammatory processes, which may end in suppuration, and hence are of the greatest importance.

The pathogenesis of these uræmic nervous symptoms may be ascribed with certainty to the retention of the constituents of the urine. Treitz and Jacksch have set up a special theory for these cases, and consider these nervous symptoms to be the products of true ammoniæmia. They assume that the ammonia from the decomposed alkaline urine is taken up by the blood, and when it has accumulated in sufficient quantities produces the manifestations in question. The possibility of absorption of ammonia has not been demonstrated as yet. However, if, according to Frerichs' theory—which has been attacked, it is true, but never confuted—it is the presence of carbonate of ammonia in the blood which causes the uræmic symptoms in chronic nephritis, there can be no reason to otherwise explain the analogous manifestations in pyelitis. It is true that the presence of carbonate of ammonia in the blood has not yet been actually proved; but when Rosenstein claims that, in experimental poisoning with ammonia, manifestations of irritation (convulsions, etc.) alone occur, while in the cases in question all the symptoms are those of pronounced depression, I think this observer goes too far in the application of the pathological experiment. For the present, at least, this question must be allowed to remain as unsettled as is the whole subject of uræmia.

Among the forms of pyelitis occurring in gynecological practice, the cases of *idiopathic* pyelitis in child-bed present a very well-marked clinical picture, which can hardly be overlooked or falsely interpreted. The disease, when it is limited to the urinary organs, begins always with a sudden onset of fever, which is usually ushered in by a more or less protracted chill; at the same time a characteristic pain is felt in the lumbar region on one or both sides. It is increased by pressure, occasionally prevents the patient from lying upon the affected side, and may also extend laterally under the false ribs. The pain always extends along the ureter downward into the region of the groin and bladder, and is occasionally indistinguishable from that of renal colic. The urine is always acid, of variable color, and is mostly quite clear when voided, except that the portions last passed may be cloudy. The specific gravity and the quantity are normal. It contains a moderate amount of albumen with temporary flocculent deposits. Microscopic examination always reveals pus in varying amounts, along with variously shaped epithelial cells from the urinary passages. In half of the cases under Kaltenbach's observation large numbers of the epithelial cells of the renal pelvis could be recognized with certainty.

The *secondary* pyelitis which occurs during the puerperal period, as a complication of grave puerperal affections, may easily be overlooked in consequence of the attention being enchained by the latter. The characteristic symptoms, however, and the condition of the urine, will enable the diagnosis to be made when the case is watched with sufficient care. In a few cases the quantity of urine appears to be diminished, probably in consequence of a temporary retention of urine.

Diagnosis.

The diagnosis of pyelitis can be made with certainty when the *characteristic epithelial cells of the renal pelvis* are found in purulent urine. The acid reaction of the urine, which is accepted by many as a diagnostic criterion, does not prove that the pus is formed in the renal pelves. The pains in the region of the kidneys, which extend along the ureters into the bladder, are also of diagnostic importance, but only in conjunction with a purulent urine. If these pains are absent, if the characteristic epithelial forms are wanting, if, in addition, the signs of a vesical affection are present, and especially if the symptoms of some affection of greater pathological importance, as, for instance, those of a grave constitutional disease, are prominently present, the pyelitis may be overlooked.

The diagnosis of pyelonephritis, which is developed in consequence of retention of urine and its ammoniacal decomposition, is often difficult. We will in these cases first of all find one of those affections of the urinary apparatus which are liable to cause retention. The urine will present no changes which are characteristic of pyelonephritis. Two groups of symptoms will, however, furnish the clue to the diagnosis, namely, the fever and the nervous symptoms. When, in connection with such an affection, chills and continued fever, with prostration of the strength and a status typhosus set in, or when vomiting occurs with a dry, crusted tongue, accompanied by diarrhœa and profuse sweats, hebetude, unconsciousness and delirium, the diagnosis of a so-called pyelonephritis parasitica may be made.

The diagnosis of pyelitis and pyelonephritis calculosa, and also its prognosis, course, duration and treatment, will be considered under the head of Nephrolithiasis.

Duration, Termination, and Prognosis.

The duration of pyelitis catarrhalis is in general not very long. The secondary form, *e. g.*, in typhoid fever, in my experience, generally disappears quickly as soon as convalescence sets in. The idiopathic pyelitis of the puerperium, in the cases observed by Kaltenbach, always ended in recovery in from five to eight days. The course of pyelonephritis due to retention and ammoniacal decomposition of the urine is also usually very rapid. In fourteen cases reported by Dickinson the duration varied from two to eighteen days. In the case of a woman, who was catheterized for retention of urine after a fracture of the thigh, death took place three days after the urine showed the first changes. Sometimes the course is more protracted. The termination does not appear to be always fatal, since we sometimes find in the bodies of persons, who have long suffered from the above-mentioned affections of the urinary passages, cicatrices which apparently resulted from the healing of small abscesses.

Treatment.

The cases of secondary pyelitis of the catarrhal variety, which are now and then observed in connection with infectious diseases, heal spontaneously with the disappearance of the original disease. The graver diphtheritic forms which occasionally occur under analogous conditions escape both diagnosis and treatment. Likewise special therapeutic measures are not required, according to my experience, for the idiopathic pyelitis of the puerperal period. With regard to the pyelonephritis, which occurs in consequence of the retention and ammoniacal decomposition of the urine, the most important part of the treatment consists in the prophylaxis. In all cases where the existing original disease demands catheterization, it follows from what was said under the head of Pathology that the greatest care must be taken to keep the catheters scrupulously clean. The reader is referred to treatises on diseases of the bladder for fuller details on this point as well as for the appropriate treatment of the vesical affections, etc. It suffices here to point out that Traube, even in apparently severe cases, obtained very good results from the injection into the bladder of solutions of acetate of lead (from half a grain to one and a half grains in four fluidounces of distilled water) and internal use of tannic acid in pill form (from one to one and a half grains at a dose every two hours). He employed both remedies on account of their antiseptic and at the same time antiphlogistic action. He ordered the tannic acid internally particularly on account of the tendency of the process to extend to the ureters and the renal pelves. The more thoroughly we succeed in preventing the ammoniacal decomposition of the urine and in stopping it if it has begun, the longer shall we be able to prevent its injurious action on the kidneys. Often, too, we may succeed, by the energetic employment of this treatment, in bringing to a standstill and even to cicatrization a commencing affection of the kidneys. Dickinson recommends the use of the mineral acids, especially sulphuric acid, in order to preserve the acidity of the urine. The internal use of carbolic acid seems to have no effect upon vesical or renal catarrh, although it is excreted in the urine. Salicylic acid might also be tried here. Like carbolic acid, it is antifermentative and antiseptic, but it may be given in much larger doses.[1]

If a catarrhal pyelitis is somewhat more protracted, which is apt to be the case particularly in the rheumatic forms, an early use of those remedies which exert an astringent action upon the blood-vessels of the mucous membrane and lessen the hypersecretion of mucus seems to be indicated. For this purpose the use of tannic acid or alum may be especially recommended (both in doses of from one and a half to four and a half grains, in powder, or half a grain of acetate of lead several times daily). The use of lead necessitates careful watching to prevent the occurrence of saturnine intoxication. At the same time constipation must be prevented by the use of mild laxatives (castor-oil, or a dose of four and a half grains of rhubarb), or of clysters. When there is severe hemorrhage, the solution of chloride of iron, in doses of from two to four drops, must be given in a mucilaginous vehicle, if the above mentioned milder astringents fail to arrest it.

---

[1] Comp. *Kolbe*, Journal f. prakt. Chemie. Bd. X. Jahrg. 1874, and *Julius Mueller*, Sitzungsberichte der Schles. Ges. f. vaterl. Cultur. 1874.

Nephrophthisis. Caseous Inflammation of the Kidneys, the Renal Pelves, and the Ureters.

Introductory Remarks. History and Literature.

Cheesy inflammation of the kidneys, the renal pelves, and the ureters was formerly, and is still by the majority of observers, ascribed to tuberculosis. A large proportion of the pathological anatomists support this view on the assumption that the origin of the isolated cheesy infiltrations from an aggregation of miliary tubercles can be demonstrated, and that it is always possible to discover small miliary nodules in the vicinity of isolated infiltrations. Without desiring to deny any positive observations, I must, nevertheless, insist that in a great number of instances neither of these assumptions proves true. Moreover, the former alone would possess any value as evidence, because the latter would not indicate the tuberculous nature of the disease. For we see true miliary tubercles appear in close proximity to the most various pathological processes, without conferring on these a tuberculous character. The manifold varieties of chronic inflammation of the lungs furnish the finest paradigms of this. The miliary tubercles that are met with in connection with them are quite accidental.

We do not know what part the miliary tubercles in the kidneys take in the development of the symptoms of general tuberculosis. An alteration of the urine does not appear to have been as yet observed in that affection. It is different, however, with the clinical history of the caseous inflammations of the kidney, renal pelvis, and ureter. We are indebted to Ammon (1833) for the first attempt to point out clearly the diagnostic points of this affection. In his masterly work, however, he impartially gives all due credit to the earlier attempts made in this direction. The following is a list of the most important authorities on this subject, which are not included in the works already enumerated on page 543.

Meckel, Handb. der path. Anatomie, 1818. II. S. 383.—*F. A. v. Ammon*, Rust's Magazin, 40. 1833. S. 500.—*Mohr*, Beiträge zur pathologischen Anatomie, Kitzingen, 1840. S. 256.—*A. Schmidtlein*, Ueber die Diagnose der Phthisis tuberc. der Harnwege. Dissert. inaug. Erlangen. 1862.—*Rilliet* et *Barthez*, Malad. des enfants. 2 edit. III. p. 852.—*Virchow*, Geschwülste. II. S. 655.—*Mosler*, Archiv der Heilkunde. 1863. S. 209.—*Rosenstein*, Berl. klin. Wochenschrift. 1865. Nr. 21.—*C. E. E. Hoffmann*, Deutsch. Archiv f. klin. Medicin. III. S. 67.—*Huber*, Deutsches Archiv f. klin. Medicin. IV. S. 609.—*Wood*, A treatise of the practice of medicine. Vol. II. Philadelphia, 1866, p. 622.

Etiology.

Nephrophthisis is met with oftener in men than in women, about in the proportion of two to one. No period of life is free from the liability to it, but the great majority of cases occur during middle age. Roberts classified 31 cases according to age. He found that of these 4 cases occurred during the first decennium of life, 5 during the second and fifth respectively, 6 during the third, 9 during the fourth, and 7 during the sixth. Isolated cases have been observed in still more advanced ages. The disease is sometimes primary, being developed first in the kidneys; sometimes in men secondary to caseous inflammation of the genitals, sometimes secondary to caseous inflammation of the deeper urinary passages, etc. The exciting cause of the affection we do not yet know. "Catching cold" plays a great rôle in the etiology, but whether justly so or not is still uncertain. A few of the patients belong to phthisical families.

Pathology.

Pathological Anatomy.

The anatomical appearances are exquisitely distinct and unmistakable.

Yellow diffuse infiltrations of greater or lesser extent are found in the renal tissue, so that oftentimes a major part of occasionally even the entire organ is occupied by these masses, and has lost all traces of its original structure. The infiltrations present an exquisite picture of the so-called *cheesy inflammation.* When the cheesy masses crumble away, the real typical picture of nephrophthisis is presented. The liquefaction of these infiltrations and the separation of the renal substance involved takes place under the form of a true putrid ulcer. The pyramids may be affected successively to the cortical substance, or vice versa. Frequently the papillæ are the first parts of the renal substance to be involved, the disease extending to them from the pelves of the kidney. Hence, in these cases the ureters and the renal pelves, or the latter alone, were the parts first affected. This mode of development is quite frequent. The changes in the mucous membrane of the renal pelvis are entirely similar to those which occur in the kidneys, only they have, of course, a superficial expansion. The kidneys may, however, be affected primarily and the renal pelvis secondarily by the softening of the cheesy infiltrations and their discharge into the renal pelvis. However this may be, the kidney in toto usually increases in size during the development of the disease, though in some rare cases it is a little smaller than normal. Its surface becomes nodular and the capsule, especially at the points which correspond with peripheral cheesy masses, is not only decidedly thickened and at times of cartilaginous consistency, but also often contains cheesy foci. When the process attains its highest grade the kidney is converted into a thick-walled sac filled with a soft, cheesy mass. This sac is incompletely divided by a few connective tissue septa—the remaining renal calyces—and forms a single cavity with the renal pelvis, which has undergone similar changes.

Sometimes the caseous inflammation begins in the mucous membrane of the bladder, and thence extends uninterruptedly through the ureters to the kidneys. In exceptional cases cheesy masses in the prostate may be the starting-point of the disease. By far most frequently, however, the primary cheesy inflammation is seated in the testicle and the epididymis, and the process is conducted to the kidney either by direct continuity of tissue —the spermatic cord, prostate, bladder and ureters being in turn involved—or discontinuously, one or all of these parts remaining unaffected. In the female sex the case is, of course, very different. Cheesy inflammation of the ovaries is an extremely rare affection, but this process is more frequently met with in the rest of the sexual apparatus, and especially in the Fallopian tubes. With regard, however, to the combination of nephrophthisis with caseous inflammation of the female genitals, it is evident, from the scanty material at our command, that though cheesy inflammation of the female urinary organs can certainly extend to the sexual organs, apparently the converse case does not occur. Usually only one kidney is diseased, and more frequently the left, a fact to which as early an observer as J. F. Meckel drew attention; sometimes both kidneys are involved, but, as a rule, one is more affected than the other. The coincident increase in the volume of the kidney has already been mentioned. This is in great part due to the distention of the renal pelvis by the dammed up urine and the collection of cheesy masses. These occasionally fill the entire ureter, the lumen of which is, moreover, already narrowed by the stiff, yellow, cheesy infiltration of its mucous membrane. In a few cases the connective tissue surrounding the ureter is found infiltrated with pus, and occasionally, when there is a simultaneous affection of the bladder, the connective tissue surrounding that organ is found diseased in a similar manner. In the latter case swelling and caseous degeneration of the neighboring lymph-glands are often met with. In the vicinity of the cheesy spots miliary tubercles are very frequently, but by no means constantly, found (vid. New Growths of the Kidney). Sometimes miliary tubercles are found at the same time in all or very many of the organs of the body. Not infrequently pulmonary phthisis and nephrophthisis exist together, and the latter has also been observed in connection with cheesy inflammation of the vertebræ.

Symptomatology.

The symptoms of cheesy pyelitis and pyelonephritis are by no means clear enough in all cases to enable us to sketch an accurate, reliable clinical picture. The symptoms which usually attract attention to the disease are those of a *chronic inflammation and ulceration in the urinary apparatus*, which sometimes present certain peculiarities. Oftentimes all symptoms are wanting.

Frequently the *urine* is altered. There are two possible cases, however, in which this is not the case—namely, when only one kidney is affected and the ureter is *plugged or compressed*, the mucous membrane below the point of obstruction being healthy; and when both kidneys are affected, but the infiltration has not yet begun to break down. The elder Meckel maintained that suppuration is a rather rare occurrence; and only on this hypothesis could his statement, that the urine is regularly secreted and is only much clearer than usual, even when both kidneys are in a state of almost total degeneration, be explained. Later observations have repeatedly controverted this statement. If we examine in detail the changes in the urine observed in nephrophthisis we will, in the first place, find that in the majority of cases the urine contains blood and pus. But as regards the time at which these abnormal ingredients appear in the urine, the individual cases vary greatly. Often the urine contains blood only in the beginning of the process; in other cases no blood is present during its entire course, but pus alone is found. In rare cases there is at first a simple albuminuria with very few epithelial cells.[1] Sometimes the consistence of the urine is so much increased in consequence of the masses of bloody mucus it contains, that it is voided with difficulty (Wood).

In the greatest number of cases the urine is acid; and only rarely, when the bladder is also diseased and the proper conditions for the decomposition of the urine exist, is it found to be ammoniacal.

---

[1] *Magnan*, Gaz. méd. 1867. Nr. 25.

The microscopical examination reveals, besides blood-corpuscles, both normal and abnormal pus-corpuscles. The latter are characterized by their irregular, often half broken up forms; even when treated with acetic acid, they do not present any distinct nuclei, but contain only small, irregular granules. In addition to these the sediment contains granular amorphous masses, the so-called detritus masses. Epithelial cells from the urinary passages are also found, and greatly swollen epithelial cells from the bladder, either isolated, or still hanging together in lamellæ. These are sometimes in a state of fatty degeneration. Tube-casts are observed when there is a synchronous diffuse nephritis. When alkaline decomposition of the urine takes place, the greater number of the cells, together with the pus-corpuscles, are transformed into a viscid mass. In a few cases of caseous pyelonephritis *elastic fibres and shreds of cast-off connective tissue* are found in the urine. They always indicate that the ulcerative process extends deep into the mucous membrane, and has attacked the tissues beneath it. A specific and pathological signification attaches itself as little to them as to the elastic fibres, etc., found in the sputum in the course of ulcerative processes of the lungs. The demonstration of small and occasionally of larger cheesy fragments, which are insoluble in acetic acid, and consist microscopically of inspissated cheesy elements, which are insoluble in acetic acid, of granular detritus and elastic fibres, is of far greater pathognomonic importance for the existence and the phthisical nature of the process in question. Besides other observers, Lebert and Vogel have particularly called attention to these masses, the latter to nodules the size of a pin's head. They have as yet been observed in no other ulcerative processes of the urinary passages than those resulting from cheesy inflammation.

Pain in the lumbar regions, which rarely radiates towards the corresponding thigh, is also a symptom of nephrophthisis. In isolated cases there is, in addition to pain, a sense of numbness, consequently disturbances of sensibility. The pains vary in character. Sometimes they are lasting and continuous, and again they occur in paroxysms, with intermissions of several days or a week. In some cases they are dull and of slight intensity; in others, on the contrary, they are exceedingly sharp and severe. Oftentimes there is no spontaneous pain whatever, but only tenderness upon pressure over the kidneys. The attacks of pain seem to depend in no small degree upon the occlusion of the ureter by the cheesy masses, which prevents the discharge of the urine and causes it to collect in the renal pelvis. As soon as the ureter becomes free, the pains diminish and the quantity of urine increases. If the urinary passages below the occluded point are healthy, the urine is free from pus while the obstruction continues, but again becomes purulent as soon as this is removed. In some cases the pains in the kidney are absent during the entire course of the disease.

In the progress of the disease the quantity of urine diminishes with the constantly progressing destruction of the secreting substance. If the mucous membrane of the bladder be also involved in the caseous inflammation, there is burning pressing pain during micturition, and now and then retention of urine. In rare cases incontinence of urine is observed (Mohr). This of course sets in, when, as in Mosler's case, a vesico-vaginal fistula is established in consequence of cheesy infiltration and destruction of the wall of the bladder. Nevertheless, as a case recorded by Huber teaches, severe strangury and vesical pains may be present, although the mucous membrane of the bladder does not participate in the cheesy inflammation; and hence, we must avoid a positive diagnosis of the localization of the process, even when the subjective local symptoms are most severe. In Huber's case there was no cheesy inflammation of the vesical mucous membrane or of the urethra, but only a marked injection of the mucous membrane of the bladder, as well as a *vessie à colonnes.* It is, indeed, possible that the altered conditions of the urine, especially the pus it contains, may cause these vesical symptoms.

The *demonstration of a renal tumor*, which, as we will see, can almost always be made in cancer of the kidney, is rarely possible in nephrophthisis. When a tumor of the kidney does occur, it very rarely attains the great size of a cancerous tumor. However, a few have been observed of the size of a child's head.

The tumors are mostly painful. In rare cases the lumbar region is found bulging outward, arched, tense, and painful. The size of the renal tumor may vary, probably in consequence of temporary occlusions of the very narrow ureter (Mosler).

This local ulcerative process reacts upon the general health. Fever is developed, which may present temporary intermissions, but is never permanently absent. In some cases there may be chills lasting for hours. Finally the fever assumes a hectic character, and night-sweats are developed. The *digestion* is impaired at the same time.

Only in rare cases does the stomach remain unaffected. Thus, Ammon reports a case of nephrophthisis in a girl, aged three and a half years, who was temporarily affected with boulimia. The sympathetic gastric manifestations so often observed in connection with other affections of the urinary apparatus, are also often met with here. Occasionally towards the end of life profuse diarrhœa sets in, which may be due to complicating intestinal affections, but may also occur without them. Purulent diarrhœa may occur when the cheesy vomicæ in the kidney open into the colon. This mode of termination is exceedingly rare, as is also that by perforation into the peritoneum. The latter was observed by Lundberg[1] in a woman thirty-four years old. The escaped matter became encapsuled, and the patient was still living one and a half years afterward.

Diagnosis.

The diagnosis of cheesy inflammation of the urinary passages is difficult. In a few cases it can be made intra vitam, with a certain degree of probability, from the concurrence of a number of the signs mentioned in the Symptomatology. For instance, the existence of this affection may be suspected when, after the extirpation of a testicle, which has undergone caseous degeneration, the symptoms of a pyelitis supervene. But if the cheesy inflammation be developed primarily in the kidneys or the urinary passages, and blood and pus are found in the urine, the correct diagnosis can only be conjectured when all other diseases which cause analogous symptoms, such as urinary calculi, stricture of the urethra, and hypertrophy of the middle lobe of the prostate, can be excluded. An examination of the prostate is absolutely necessary in all cases of purulent urine, and might here also often give a clue to the diagnosis. A nodular, painful prostate, in connection with the presence of blood and pus in the urine, points with a certain degree of probability to this organ as the starting-point of the caseous process. The appearance of cheesy masses in the urine is of the greatest importance in the diagnosis, as has already been explained. Whether and to what extent the kidneys, the renal pelves and the ureters are implicated in the cheesy inflammation, are questions which it is very difficult and at times impossible to answer. The following are the diagnostic points of value here:

---

[1] Schmidt's Jahrb. 91. S. 74.

In general, the kidneys, the renal pelves, and the ureters, or, at least, their upper portions, are affected far more frequently and more severely than the bladder. *A priori*, therefore, we may assume that the parts in question are always involved whenever a caseous inflammation of the urinary passage exists. This *à priori* assumption, however, does not secure a sure foundation until a tumor of the kidney can be demonstrated, and the colicky pains, which also occur in connection with other forms of pyelitis, set in. The early loss of the strength and all the signs of a rapidly progressing marasmus, in addition to the fever, will excite the suspicion of a phthisical affection.

Duration, Course, Prognosis.

It is very difficult to accurately determine the duration of cheesy nephritis, because the time of its commencement can, at best, be only imperfectly determined. The interval between the beginning of the characteristic symptoms and the fatal termination is rarely longer than a year, and frequently is not longer than a few months. In a few cases the disease lasts two, and, very rarely, three years.

The *prognosis* is bad. The hope of Roberts, that, when only one kidney is involved in the disease, the cheesy masses may be discharged through the renal pelvis, although it is based upon a case reported by Bennet, is not on that account well founded, because in that case there may have been simply an abscess with inspissated contents. Even the early extirpation of tumors of the testicle, which are produced by infiltration with cheesy masses, does not appear to arrest the advance or prevent the development of a similar process in the kidneys.

Treatment.

All treatment is apparently useless. It must be conducted on the principles which are usually laid down for the treatment of cheesy inflammatory processes elsewhere. A nutritious diet, combined with mild stimulants, and cod-liver oil in combination with iron, are especially indicated. At the same time the preparations of iodine may be cautiously employed, especially in combination with iron, and eventually brine baths may be tried. When the pain is severe, narcotics cannot be dispensed with. The remaining symptoms are to be combated by the measures appropriate to them, particularly the profuse diarrhœa, which greatly accelerates the fatal termination.

Peri- and Paranephritis.

Definition, History, and Literature.

I consider in this section the inflammatory and suppurative processes which take place in the tough fibrous capsule directly enclosing the kidney, and in the loose connective and adipose tissue surrounding it. The kidney is imbedded in a loose mass of connective tissue, more or less abundantly filled with fat, in front of which the peritoneum descends, and which is connected with the firm fibrous capsule by fine, easily torn fasciculi. Inflammatory processes in the renal capsule do not attain the dignity of independent pathological processes, while those localized in the connective tissue surrounding the kidneys, which is directly continuous with the connective tissue contained in the retroperitoneal, abdominal, and pelvic spaces, are described as *peri-* and *paranephritis.* The peri- and paranephritic inflammations are mostly combined with each other, and the practitioner will hardly ever be able to separate them *intra vitam.* I shall employ, therefore, in the course of the following description, for the sake of brevity, the term "perinephritis" to designate both these pathological processes.

It was well known to the earlier observers that suppurative processes resulting in the formation of abscesses occur in the connective tissue surrounding the kidneys, but the first connected description of this rather rare, but yet practically very important affection, was given by Rayer in his "Maladies des reins," T. III. p. 243, Paris. 1839. After him this chapter was elaborated by Féron, "De la périnéphrite primitive," Thèse, Paris. 1860. Parmentier, "Union médicale," vol. XV. 1862. Lemoine, Ibid. vol. XVIII. 1863. Hallé, "Des phlegmons périnéphritiques," Thèse, Paris. 1863. Guérin, "Gaz. des hôpit." 1865. With the help of most of these articles and of still other material, as well as of numerous observations of his own, Trousseau was enabled to furnish in the third volume of his "Medical Clinic" a very careful, clear, and instructive account of the affection, with especial reference to the symptomatology and therapeutics. Of late years the number of cases recorded have materially increased. Duffin[1] alone has collected twenty-six cases.

Etiology.

The perinephritic inflammation is far oftener *secondary* than *primary*. *The causes* are partly of a *general*, partly of a *local* nature. What is at present known of the etiology is contained in the following paragraphs:

In the first place, a number of cases are due to injuries. First among these must be mentioned *actual wounds* of the kidney, where blood and urine are forced into the connective tissue surrounding it, and excite perinephritic abscesses. In the great majority of these cases, however, the inflammation of the perinephritic connective tissue is excited by *contusions*. Sometimes forcible movements of the body, such as the severe jarring consequent on long-continued riding, or on driving in a jolting wagon, are sufficient to cause contusions. Of course, the assumption that in these cases the irritation is confined to the connective tissue surrounding the kidney is doubtful, though it is supported by a number of recorded facts. Hallé maintains that a sudden chilling of the overheated body is a concurrent factor in the etiology of all these cases. This, however, does not make the subject any clearer. Far more easily explained are the cases where a veritable inflammation of the connective tissue surrounding the kidney is developed after great bodily exertions, the lifting of heavy weights, etc., in consequence of a rupture of the tissue in the lumbar region. This accident is usually revealed by the immediate occurrence of an intense pain.

---

[1] Med. Times and Gazette, 1872. vol. ii.

Chronic inflammations of the kidney and of the *renal pelvis* extend not unfrequently to the tissues surrounding the kidney. Chronic pyelitis and pyelonephritis, and particularly those of calculous origin, are especially apt to cause perinephritis in this way. Echinococci, when developed in the connective tissue in the vicinity of the kidney, may also give rise to inflammatory manifestations. In rare cases *abscesses in the iliopsoas*, consequent to diseases of the vertebræ, secondarily involve the perinephritic connective tissue; but, as a rule, the firm fibrous sheath of the muscle confines the process to the muscle itself. At the head of the other causes which incite secondary perinephritic abscesses must be placed the *extension of inflammatory processes* from the cellular tissue surrounding the lower part of the uterus and the upper part of the vagina, and also from the pelvic connective tissue, *upward to the retroperitoneal perinephritic connective tissue.* These inflammatory processes are predominantly of a puerperal nature, but are developed also from various causes in the non-puerperal state, especially after operations upon the collum uteri, etc. Finally, they may also arise from so-called internal causes, such as idiopathic affections of the collum uteri, exposure to cold, etc. In *men*, *all affections which involve an irritation of the testicles and of the spermatic cord may excite inflammation of the pericystic and the pelvic connective tissue in general*, which may extend to the perinephritic connective tissue, and there result in the formation of abscesses. Operations upon the rectum may have similar effects, *e. g.*, extirpation of the rectum, even when the peritoneum is not perforated. Koenig[1] relates an interesting case of this kind. The patient died of peritonitis, which was secondary to a spreading septic phlegmon in the subserous tissue. This had travelled upward in the pelvic cellular tissue along the posterior wall of the rectum, and had spread itself out in the perinephritic connective tissue as far as the colon descendens.

The occasional occurrence of *secondary* perinephritis in connection with ileotyphus, exanthematic typhus, and variola must also be borne in mind. It is, however, a rare complication. E. Wagner reports a case of purulent paranephritis, along with pyæmic abscesses in other situations, that he saw during the great variola epidemic at Leipsic in 1870–71. Rosenstein observed a case of bilateral perinephritis, consequent to typhus, although the affection is usually limited exclusively to one side. In addition to the causes already enumerated, *taking cold* must be regarded as the active cause of perinephritis in a certain number of cases where the disease is primary. Although we have many reasons to mistrust this much-abused cause of disease, it is at least certain that, in some isolated cases, the first symptoms of a developing perinephritis are experienced directly after a sudden and decided chilling of the body.

Concerning the *individual predispositions* to the disease we know very little. We cannot explain why exposure to cold, in certain cases, causes inflammation in this locality; why diffuse perinephritis of the worst kind is often developed after simple wounds of the collum uteri. Since the exciting cause of perinephritis is most frequently an injury of some sort, it necessarily follows that it is especially an affection of adult age, and that it is more frequent in men than in women. In childhood, perinephritis is extremely rare. Rosenstein could not find a single reported instance of the affection in children. I have, however, learned the particulars of a case that occurred in the practice of Loeb. The patient, a boy six years of age, received a severe contusion of the left lumbar region, which caused paranephritis. The contents of the abscesses were discharged partly into the colon, partly through an external incision. The recovery was complete. The duration of the sickness was about seven weeks.[1] Trousseau's supposition, that *pure neuralgic affections of the urogenital organs* may give rise to the development of perinephritic abscess, does not seem to be supported by the evidence adduced by this observer.

---

[1] Volkmann's Sammlung klinischer Vorträge. Nr. 71.

Pathology.

Pathological Anatomy.

The autopsy in suppurative nephritis discloses a collection of pus in the capsula adiposa of the kidney and the loose connective tissue surrounding it; a true abscess. The pus is mostly yellowish and thick, and mingled with shreds of necrotic connective tissue. In consequence of admixture with blood, it often has the different tints of the more or less metamorphosed coloring matter of the blood. The initial stages of the process rarely come under observation at the post-mortem table. The collections of pus spread themselves out in those directions in which they meet with the least resistance, particularly along the meshes of the retroperitoneal connective tissue, and extend sometimes from the lower surface of the liver or spleen to the iliac fossa. The resistance of the neighboring portions of the peritoneum increases with the advance of the inflammatory process; this membrane becomes thickened, and adhesions are developed which render its perforation more difficult.

In the beginning the cavity of the abscess is bounded by ragged walls, which later on become smooth, and are partly grayish red, partly shining white in color. When the pus burrows into the neighboring parts the abscess assumes an irregular form. When it has existed for a long time, it may burrow into the neighboring muscles—the m. quadratus lumborum, the m. psoas, and the iliacus. The abscess never undergoes resolution, but constantly increases in size, and may finally perforate the peritoneum and empty itself into the peritoneal cavity. In this case, in addition to the perinephritic abscess, the section will reveal a recent peritonitis, with more or less abundant purulent masses in the abdominal cavity. In other cases the abscess makes its way outwards, either directly or indirectly, by first rupturing into other organs. Most frequently these perinephritic abscesses burrow backward and outward, and open in the lumbar region. We then find, as a rule, suppurating cavities of considerable extent which communicate with the exterior by tortuous fistulous openings between the muscles. Much less frequently the abscess obtains an outlet for itself by perforating the wall of the ascending or descending colon, according as it is situated on the right or left side. Even in this case the pus is often inodorous. A feculent smell of the pus is by no means a proof that a communication exists between the intestines and the abscess, because it is oftentimes present where there is no such communication. Fecal masses are rarely found in the pus, even when there is a communication between the intestine and the abscess, because the opening is so constituted that it permits the passage of pus into the intestine, but does not allow the escape of fæces into the cavity of the abscess. There are exceptions to this rule, however. Again, gravitation of the pus along the vertebral column is not unfrequently observed. In these cases the abscesses resemble the ordinary congestive abscesses which start from the vertebral column, and they make their appearance upon the inner side of the thigh at the femoral ring, or in the anal fold. In very rare cases the perinephritic abscess opens into the bronchi, and the pus is discharged by expectoration. When the abscess runs a very chronic course we find at the autopsy a dense, fibrous rind surrounding the kidney. Cases of this kind are rare. They are most frequently observed in connection with pyelitis calculosa. Here, as in all cases of paranephritis, which are secondary to diseases of the kidney, *e. g.*, phthisis renalis and pyelonephritis, the capsula adiposa disappears, and in its place a tough, rind-like connective tissue appears. Apart from the primary changes in the kidneys, which secondarily produce perinephritis, the phlegmonous inflammation of the perinephritic tissue exerts an unfavorable influence upon the renal substance, and excites in it more or less marked disturbances of nutrition. The epithelial cells are often in the condition of cloudy swelling. Sometimes even necrosis of the renal substance, to a greater or less extent, takes place. In other cases abscesses are found in the renal substance separated by the capsule of the kidney from the perinephritic abscesses. In other cases again the kidney appears very dense and firm, and is at times so compressed that its size is reduced to a minimum. However, the markings of the kidney are usually distinctly recognizable.

---

[1] Jahrb. f. Kinderheilkunde. Neue Folge, VIII. S. 197.

We have thus far been describing the appearances presented by primary perinephritis, and it now remains for us to examine more closely the anatomical extension of the inflammatory process from the pelvic cellular tissue to the retroperitoneal, subserous, perinephritic connective tissue. Let us consider first the most frequent form, *the puerperal perinephritis*, which complicates puerperal inflammation of the pelvic cellular tissue. This results most frequently from the development of an abscess in the connective tissue of the broad ligament in the vicinity of the Fallopian tube and the ovaries. The abscess insinuates itself between the peritoneum and the psoas and iliacus muscles, and raises the former from the latter. The accumulations of pus which develop themselves about the lower part of the womb in the immediate vicinity of the cervix follow a different course. The first fill up the lower subperitoneal connective tissue of the true pelvis, and thence accompany the round ligament of the uterus, the lig. Poupartii, and the inguinal canal. From this situation they extend backward through the fossa iliaca and then upward. Also in connection with affections of the bladder, of the spermatic cord, and of the rectum, and especially after operation upon these organs, inflammatory processes may be set up in the pelvic cellular tissue, and extend through this tissue into the subserous tissue in the neighborhood of the kidneys. We have to deal here with a septic phlegmon in the perinephritic connective tissue. It is discolored, of a dirty yellowish color, and presents here and there collections of ichorous pus. It is a true phlegmone para- et perinephritica,—we have to deal with the process which has been designated sometimes as cloudy serous infiltration (Buhl), sometimes as sero-purulent œdema (Pirogoff).

Symptomatology.

The signs of perinephritis appear in the different cases with very various degrees of clearness and distinctness. In the cases which are secondary to inflammation in the pelvic cellular tissue, *e. g.*, a puerperal parametritis, the constitutional symptoms are so prominent, and the cases are so rapidly fatal, that the symptoms produced by the secondary perinephritis, if indeed there are any, are entirely overlooked. Nevertheless, even in these cases marked painfulness in the region of the kidneys is often observed. Recovery is possible in some of these cases when the contents of the abscesses are discharged. Kaltenbach[1] cites an instance of it. A healthy woman, on the fifth day after delivery began to suffer from severe lumbar pain accompanied by fever, and at the same time from pain in both inguinal regions. The symptoms of pyelitis were not present. In the left ligamentum latum a painful swelling was discovered. In the second week pleuritis set in, and five weeks after the delivery two abscesses were found in the region of the kidneys, which were opened and discharged great quantities of pus. These cases, however, are at all events rare.

The symptoms of *primary* purulent perinephritis and of those forms which are secondary to the various renal diseases, and especially to pyelonephritis calculosa, are far more pronounced. Nevertheless, even in a certain number of these cases, the perinephritic abscesses escape the notice of the physician in consequence of the local manifestations, such as the pain, being overshadowed not only by the constitutional disturbances, but also particularly by more severe pains elsewhere. *The symptoms of primary suppurative perinephritis* manifest themselves, as a rule, as follows: One of the most important initial symptoms is the *pain.* It sets in suddenly after exposure to any of the already mentioned causes, is deep-seated, dull or pricking. The pain is increased by pressure, especially by deep pressure. This pain, which marks the beginning of the disease, usually continues throughout its whole course with uniform or increasing intensity. In other cases the severity of the pains diminishes temporarily, or they disappear entirely for days, weeks, or even months, encouraging a delusive hope that recovery has taken place. At last, however, they set in afresh, either with or without apparent cause, and are oftentimes severer than before. In addition to the pain, *certain general symptoms* are present from the outset of the affection, and indicate that the pain depends on some important organic disturbance. The patients have a continued fever of considerable intensity ; the evening temperature usually reaches 103$\frac{1}{10}$° F. and may be higher, and in the morning there are slight remissions of about $\frac{9}{10}$° F. The rigors are followed by fever and pespiration. At the same time the *digestion* suffers, the appetite becomes poorer, and the taste slimy ; the febrile attacks are often accompanied by vomiting, and the constipation is obstinate and can only be relieved by medicaments, and even then often only incompletely. Sometimes a considerable period, during which the patient complains of a feeling of debility, intervenes between the time of exposure to the exciting cause, and the development of the disease in the above mentioned manner. When the suppurative inflammation of the perinephritic connective tissue is consecutive to nephrolithiasis, it is preceded by the symptom which will be described in the chapter on Renal Concretions. When the affection has lasted a longer or shorter time a new symptom is added to those already described, namely, a *tumor*. The appearance of this symptom is preceded by a constantly increasing sensitiveness and a sensation of increasing tension. An oval or rounded swelling is next discovered, usually in the lumbar region but sometimes a little lower down, about the situation of the S. romanum. The suspicion that, in the latter case, the swelling might be due to accumulation of fæces, disappears if a thorough evacuation of the lower part of the colon by means of clysters does not remove the tumor, and percussion over the colon does not reveal the presence of masses of fæces. When the tumor is situated on the right side, the fact that its position is not affected by the respiratory movements shows that it is not connected with the liver. As the swelling increases, the shallow depression, which is visible under normal circumstances in the lumbar region, is filled up. Oftentimes a pseudo-erysipelas of the corresponding portions of the skin sets in, which may be associated with an œdematous swelling, extending in some cases over the entire back.

---

[1] Archiv. f. Gynaek. III. S. 24.

Whilst the process is advancing in this manner, a more or less distinct and deep-seated fluctuation can now and then be demonstrated. It is by no means easy, nor is it always possible, to do this. The pus lies deep, and long practice is required for quick perception of the feeling of fluctuation. At the same time the febrile manifestations increase, and particularly, as the suppuration increases, true rigors often occur. Moreover, in the course of this purulent perinephritis a feeling of numbness is experienced in the corresponding lower extremity, which is also kept in the flexed position. As soon as distinct fluctuation is felt, however slight it may be, it is time to evacuate the deep-seated collection of pus. If this be not done early enough, the pus clears a passage for itself by burrowing in the direction where the least resistance is presented to it. Experience teaches that the perinephritic abscess, left to itself, opens most frequently in the lumbar region. The collection of pus becomes encapsuled, and then a circumscribed portion of the skin, which is the seat of the greatest pain, assumes a more and more arched form. This spot becomes constantly more doughy and softer, fluctuates, and at the same time becomes more pointed. Finally a perforation occurs spontaneously, or a puncture with a lancet suffices to secure an exit for the pus. Such perinephritic phlegmons may attain a very great size before the pus breaks through into any of the localities that will soon be mentioned. As much as one or two quarts of pus may escape. I have already mentioned above that the pus may be very offensive, even when the abscess does not communicate with the intestine. However, this is exceptional. The improvement in the general condition, which immediately follows the evacuation of one of these abscesses, is very marked. The temperature sinks at once to the normal point, the pseudo-erysipelas disappears, and the bowels, which up to this time were obstinately constipated, sometimes so much so that purgatives produced no effect, become regular.

When the abscess is not evacuated through a spontaneous opening in the lumbar region, or through a free incision made as soon as fluctuation is discovered, the pus burrows in some other direction. In many cases it sinks into the iliac fossa, and the patients complain of severe pain throughout its entire extent. A soft, fluctuating swelling then appears above or below Poupart's ligament, and the pus breaks through spontaneously, or is emptied by incision. Sometimes the bursa subiliaca is involved in the suppurative process, and then the pus gets into the hip-joint, because this synovial sac almost always communicates with the cavity of the joint. The symptoms assume a different form when the pus escapes into the intestine. In that case great quantities of pus may pass off with the stools. Externally *emphysema*, involving the entire extent of the back, is sometimes developed. Trousseau has observed this twice. In both cases the abscesses were opened, and offensive gases escaped with the pus. In one patient the communication of the abscess with the intestine was demonstrated by the presence of pus in the stools, and also by the mixture of feculent masses with the pus escaping through the incision. When the pus sinks into the true pelvis, it is often discharged through a spontaneous opening into the vagina or the bladder. When the perinephritic abscess perforates the diaphragm and opens into the lung and bronchi, as it has been known to do in some very rare cases, its evacuation is effected by means of the sudden and immediate expectoration of large quantities of pus.

The rupture into the peritoneal cavity is the rarest of all the terminations of perinephritic abscess. The symptoms that arise in that case are those of an extremely acute peritonitis. Vomiting sets in, the belly becomes extremely painful, the pulse becomes very frequent and thready, and the signs of the most marked collapse precede death. The urine presents nothing characteristic in suppurative perinephritis, except in the cases where the affection is secondary to a pyelonephritis calculosa. It possesses the characters of a purely febrile urine. It is scanty, darker, even brownish red in color, and contains more or less urates.

Specific gravity 1015–1020; reaction acid; no albumen; no casts. In some rare cases of perinephritis the inflammatory process undergoes resolution, all the symptoms—pain, fever, and tumor—retrograding and gradually disappearing.

Complications and Sequelæ.

In a certain number of cases inflammatory processes are developed in other localities, which oftentimes seem to be connected with the perinephritis, although this connection is not directly demonstrable, *e. g.*, pleurisy upon the affected side. As these occur in individuals who are already debilitated by the existing suppuration, they have a tendency to produce rapidly increasing exudations. Moreover, nephritis is now and then a complication of purulent perinephritis, as well as of other abscesses. Both complications render the prognosis very much more unfavorable. A very instructive case, which illustrates both these complications, has been recently described by Rahn.[1]

In this case an empyema of the left side was added to a primary perinephritis of the same side without it being possible to trace any direct connection between the two. After the perinephritic abscess had been emptied, the increasing dyspnœa made thoracentesis also necessary, and the operation gave exit to an ichorous purulent exudation. To the symptoms of these two affections the signs of an acute peritonitis were added a few days before death. At the same time the urine became blackish brown in color, and examination revealed the presence of large quantities of albumen, tube-casts, and blood, which had never been present before.

Pneumonia of the affected side has also been met with as a complication of perinephritis suppurativa. If the perinephritic abscess burrow downward it may give rise to abscesses in the M. psoas by the extension of the inflammation to that muscle.

Diagnosis.

I have already pointed out above that the symptoms of perinephritis consist of pain, certain general disturbances, especially fever, and a tumor in the region of the kidneys, which is due to the formation of pus. The diagnosis must consequently be based upon the presence of these symptoms. In certain cases, nevertheless, the diagnosis is very difficult. The pain itself is, in general, but slightly characteristic in the beginning of the disease. When, however, the pain is localized by the patient deeply in the tissues, is unilateral, and follows one of the known causes of the disease, such as a contusion or a severe strain, it furnishes at least a hint to the diagnosis. This will be supported by the presence of another weighty symptom: the constitutional disturbances, especially the fever, which does not accompany other painful affections in the lumbar region. Neuralgic affections, as well as muscular rheumatism of the lumbar region, run their course without elevation of temperature, or the latter, at most, with a slight ephemeral fever. Oftentimes, after the affection has existed for some time with fever and pain, an almost complete cessation of all the symptoms sets in, which may easily lead to error in the diagnosis, and may be ominous for the termination of the disease in consequence of the neglect of the necessary treatment induced by the apparent recovery. The differential diagnosis from other affections of the kidney—for instance, from hydronephrosis, echinococci, malignant new growths, and especially from pyelitis and pyelonephritis—may be difficult, because all these renal diseases lead to the formation of a tumor of the kidney. As the diagnostic points will be discussed in the description of each of these affections, a brief recapitulation of the most important of these differential points must suffice at this place. In hydronephrosis and in echinococcus the tumor is usually painless; and, moreover, these diseases are not accompanied by fever, except when complicated with other affections. In hydronephrosis a fluctuating nodular tumor of the kidney is frequently felt. In carcinoma of the kidney a pseudo-fluctuation is perceived in rare cases, when the tumors are very soft; usually the tumors are irregularly nodular, of very firm consistency, without fluctuation, and exceedingly painful, and are often accompanied by hæmaturia. Here, also, fever is absent. Moreover, all these tumors of the kidney almost invariably extend in the process of development forward toward the cavum peritonei, while the great majority of perinephritic abscesses are found by far most frequently in the lumbar region, on the dorsal surface of the body. Much more difficult may be the differentiation of perinephritic abscesses from pyelitis and pyelonephritis, which are, indeed, often complicated by perinephritis. Fever, pain, and tumor, are all present in these cases; the condition of the urine, however, is diagnostic. This is commonly normal in perinephritis, for the abundant urates, which are found in this as in every febrile urine, cannot come into consideration; whilst in pyelitis and pyelonephritis, provided the ureter is not obstructed, a purulent sediment is always found in the urine. An accurate consideration of the etiology and of the mode of development of the disease will also afford a protection against error. Moreover, the œdema of the skin, provided it be present at all, is a diagnostic point for perinephritis, for it is never a symptom of pyelitis. Superficial phlegmons of the lumbar region should never be confounded with perinephritic suppurations. They run a more rapid course, and in consequence of the superficial position of the pus the skin reddens early, and distinct fluctuation is easily perceived after a short time.

---

[1] Inaugural-Dissertation. Berlin. 1873.

The flexed position of the thigh, which is similar to the position assumed when there is inflammation of the M. psoas, may readily excite the suspicion that this muscle is affected, and the differential diagnosis may be beset with difficulties. The adipose tissue surrounding the kidney lies on this muscle, and a sharp differentiation of the two processes *intra vitam* is often no easy task. In the case reported by Rahn, that has been already mentioned, the two following points decided the diagnosis in favor of a perinephritic abscess: first, the circumstance that flexion of the thigh did not excite any decided expressions of pain on the part of the patient; and, secondly, the fact that pressure over the regio renalis was extremely painful. In psoitis exactly the opposite would be the case; here it is exceedingly difficult to flex the thigh, while pressure upon the regio renalis produces no pain. Trousseau, while speaking of the diagnosis of perinephritic abscesses, warns us to bear in mind, while examining a tumor in the lumbar region, that, at the very spot where the tumor usually projects, the variety of hernia to which J. L. Petit has given his name also occurs. He had in mind then a case in which a mistake would have been made if the surgeon had not made an attempt at reduction before proceeding with the contemplated incision. In such rare cases, however, the symptoms of acute strangulation, which would at once point to the diagnosis, would never be absent.

Duration, Termination, and Prognosis.

Primary perinephritis usually runs a rapid course. Frequently the affection ends favorably within a few weeks, when it is recognized in the beginning and treated properly, and an exit for the pus is secured at the proper time. The prognosis in such cases can, in general, be considered favorable. The wound usually closes after evacuation of the abscess in simple, uncomplicated cases.

In a few cases recovery appears to take place without suppuration. Lebert describes a case of this sort :[1] a distinct swelling existed in the right renal region, with excessive painfulness and subsequent paresis of the lower extremity. The inflammation was apparently situated in the connective tissue behind the kidney. After repeated applications of leeches, inunctions with mercurial ointment, and, later on, the use of a vesicant, resolution and complete recovery took place. The longer, however, the process is protracted in the primary cases, the more unfavorable becomes the prognosis. This is partly because the pus then burrows in various directions, and, by opening into important organs, may lead to a fatal termination, and partly because the long-continued fever and the protracted suppuration not only undermine the strength of the patient, but also excite various dangerous complications. If the pus be not evacuated at the proper time, the fever sometimes rather rapidly assumes the character of a febris hectica, with colliquative discharges. Later on a typhoid condition is developed, and death is preceded by coma and delirium. When, however, the abscess in the perinephritic tissue opens spontaneously, after it has undermined the muscles in different directions, a long interval will elapse in the most favorable cases before the sinuous, irregular, fistulous canals will close. Even after rupture of the abscesses into the colon and the bronchi, recovery has several times been known to occur. The prognosis is, however, always very doubtful when the pathological processes are so complicated. The prognosis is absolutely bad when rupture into the peritoneal sac takes place.

---

[1] Virchow's Archiv. XIII. S. 532.

In the secondary perinephritis which follows puerperal processes or infectious diseases, the prognosis is almost always unfavorable on account of the gravity of the original disease; it is not much better in the perinephritis which is secondary to calculous pyelonephritis.

Treatment.

Although the treatment of primary perinephritic abscesses properly belongs entirely to the department of surgery, still the physician must be thoroughly acquainted with the rules that experience has sanctioned for it. The disease will come under his hands on account of its seat and the constitutional disturbances connected with it, and he must not allow himself to be guilty of any sins of omission. In the first place, the energetic application of cold in the form of ice-bags or ice-cold compresses, as well as absolute rest, are to be recommended, especially when the disease is due to traumatic influences. The bowels must be emptied by means of clysters. The length of time during which the employment of cold must be continued will depend on the pain, though in a certain number of cases, where the patients are intelligent and observant, the application of ice may be continued as long as it causes a sensation of comfort. Should the pain moderate, absolute rest must still be enforced for several days, until the pains and fever are entirely over. If in spite of these measures a tumor forms deep in the tissues, we may attempt to bring about resolution by the use of mercurial preparations, especially by inunctions with gray ointment, while at the same time the pains are controlled by morphia and sleep is induced by chloral hydrate. If the redness of the skin has already appeared, the gray ointment will be of no further use.

Poultices are then indicated, and, as soon as the first traces of fluctuation are perceived, the abscess must be opened. The opening is best made with the knife, and should be sufficiently large to allow the free escape of the pus. The incision in the skin must be longer than in the deeper tissues, to enable the pus to flow out readily. In making the opening, the different layers of the tissues should be divided in turn, all divided arteries should be secured, and in order to avoid all bleeding the division of the several layers should be made upon the grooved director. The incision is at all events preferable to opening by escharotics, which is a tedious and painful procedure, does not protect against hemorrhage, and is here quite useless, since it is not necessary to produce adhesions of the abdominal walls to the deeper parts, in order to prevent the entrance of pus, etc., into the peritoneal sac. Evacuation with the trocar, or by means of Chassaignac's drainage, possesses no advantage over the knife. The trocar does not empty the sac completely, and the drainage keeps up a long continued suppuration in consequence of the irritation it produces. The remaining surgical treatment, especially that of casual burrowing of pus and congestive abscesses, must conform to the rules of general therapeutics. The internal medication must be symptomatic and especially sustaining. At the same time the fever must be kept as much as possible within bounds, by the use of preparations of quinine and quinine itself. The complications are to be treated according to the special indications which they present.

The Degenerative Processes of the Kidneys.

Cloudy Swelling and Fatty Degeneration of the Kidneys.

LITERATURE:—The works mentioned upon page 543 and *Geo. Johnson*, Brit. Med. Journ. 1873. (Schmidt's Jahrbb. 161. S. 258.)

Etiology.

Experience has shown that the causes of cloudy swelling of the kidneys, which is observed as a result of granular (parenchymatous) degeneration of the epithelial structures, as well as of fatty degeneration of the secreting elements proper—both of which conditions differ from each other only in degree and are not to be confounded with fatty infiltration—are to be referred to a variety of pathological processes which possess only this feature in common, namely, that they give rise to disturbances of nutrition in either a few or many organs. These disturbances, moreover, may affect the kidneys alone or even only portions of them.

Among the *general causes* (changes in or diminution of the entire mass of the blood) may be enumerated the following: the infectious diseases, the pyæmic and septic processes, extensive phlegmonous processes, the acute exanthemata (especially variola), tuberculosis miliaris acuta, rheumatismus articularis acutus, etc., as well as many *poisons*, such as *phosphorus* in particular, sulphuric acid, carbonic oxide, and others; further, extensive burns of the skin (Wertheim), and finally, the so-called progressive pernicious anæmia (Biermer, Gusserow, Immermann).

To the *local* causes (local hinderance to the blood supply) belong: venous stasis and induration of the kidneys; inflammatory processes in the kidneys, namely, interstitial diffuse nephritis and circumscribed forms of inflammation, as well as, finally, embolic wedge-shaped infarctions with granular and fatty degeneration, following embolism of the arteries with complete occlusion of the blood-vessels. (Upon embolic infarction of the kidney, compare page 605.)

Pathology.

Pathological Anatomy.

The process chiefly affects the cortical substance, most of all, the convoluted portions of the uriniferous tubules. It is particularly in the acute processes that the epithelial cells become swollen and so give rise to a slight swelling of the organ *in toto;* the portions which have undergone this change appear anæmic and of a grayish yellow color, whilst the pyramids, which for the most part have escaped this change, appear dark red in consequence of the collateral fluxion. The epithelia of cloudy portions of the tubules appear at first homogeneous, highly refractive, and shining; later on they are finely granular, and consist finally of a uniform mass, in which can be distinguished finer (albuminous) and coarser fatty granules. The more intensively and extensively the cause acts, in so much the more marked degree shall we find the fat metamorphosis, the most pronounced degrees being encountered in phosphorus poisoning. The epithelial cells become disintegrated and fill up the lumen of the uriniferous tubules. The acute septic processes bring about very early a rapid destruction of the epithelia and softening of the entire organ. Very often so-called tube-casts are found in the uriniferous tubules.

Symptomatology.

While referring my readers, for more complete details, to those sections of this work which treat of the fundamental processes causing granular and fatty degeneration (specified on page 603), I will here introduce two points only, *to wit*, the occurrence of tube-casts in the urine, and the occurrence of albuminuria. In case of fatty degeneration of the epithelial cells, the casts are covered with granular masses and fat-drops. Albuminuria is very frequent, but is also not constant. It only occurs in so far as its fundamental causes are present, viz., changes in the walls of the vessels and modifications in the degree of blood-pressure.

Leaving the question of the diagnosis, the duration, the course, termination, and treatment to be studied in connection with the description of the fundamental diseases. I will here briefly and in a supplementary manner consider

Hemorrhagic Infarction of the Kidneys,

so far as this leads to early fatty alteration of the epithelium of the urinary tubules, as the result of an arrest of the supply of arterial blood.

Compare *Virchow*, Gesammelte Abhandlungen.—*Cohnheim*, Embolische Prozesse. 1872. Berlin.—*von Recklinghausen*, Virchow's Arch. XX. p. 205.

Etiology.

This is not the place to give a more minute description of the general method of origin of the hemorrhagic infarctions which arise from embolisms. Cohnheim's admirable experimental investigations have thrown great light on this subject. The conclusions which this observer has drawn from his experiments on the frog's tongue, with regard to the pathology of infarctions, have most satisfactorily cleared up numerous hitherto entirely incomprehensible points, for the explanation of which the term infarction has too often been called into requisition. It may suffice here to adduce the most important points bearing on infarction of the kidney. The fundamental condition on which it depends is obstruction of a branch of the renal artery. Infarctions very seldom occur after obstruction of the main trunk of the renal artery (see p. 608). The renal artery fulfills one of the first conditions for the occurrence of an infarction, inasmuch as it very completely represents a terminal artery—that is to say, the injection of a branch of the renal artery, before it enters the hilus, only fills the territory directly supplied by this branch, and only very few and small arterial anastomoses are found to exist between occasional little branches of the capsular and renal arteries. These would not suffice, in case of obstruction of a branch of the renal artery, to produce collateral circulation. Having taken it for granted that obstruction of such a branch of the renal artery has taken place, and knowing that the renal veins have no valves, we shall have no difficulty in conceiving how infarction of the kidney results, in accordance with the views deduced from experimental observations concerning the processes that obtain during the formation of an infarction. Instead of infarction of a portion of the kidney supplied by such a terminal artery, however, necrosis of that portion may take place (compare p. 608 *et seq.*), and a certain series of conditions must be fulfilled if an infarction is to be developed before necrosis of the kidney tissue supervenes. These conditions consist of a distinct, backward-flowing, venous current, and an actual total obstruction, by means of the thrombus, of the arterial branch supplying the part in question. Whatever arrests or hinders the venous reflux, or renders it more difficult, will also hinder the forming of an infarction and favor necrosis of tissue. For all the details of this process I refer the reader to Cohnheim's great work. All that could be done here was to give the most general outline.

Aside from obstructions of the renal artery through embolism, the development of infarctions may also be occasioned by wounding of the renal artery. Von Recklinghausen describes such a case. The subject was a boy thirteen years of age, who died eight days after a fall from a considerable height. The renal artery, as well as one of its twigs, showed a circular rent and the formation of a thrombus.

Pathology.

Pathological Anatomy.

Hemorrhagic infarction of the kidney, in its fresh condition, presents itself as a blackish red, tense wedge, sharply bounded on three sides, and projecting a little beyond the level of the neighboring parts. The base of this wedge is directed towards the periphery, the apex towards the centre. After a short time it begins to change color, soon assuming an intensely yellow hue. At the same time the infarction becomes somewhat more voluminous, and the epithelium undergoes fatty degeneration. After a longer period shrinking of the infarction takes place, the wedge-shaped collection grows narrower, and dense connective tissue takes the place of the renal cortex.

Symptomatology.

Hemorrhagic infarctions of the kidney give rise to no symptoms during life. There are but very rare exceptions to this rule. Traube has described such a case. A patient who was suffering from insufficiency of the aortic valve, who had felt quite well during the evening, being particularly free from pain, was suddenly wakened at night by a severe pain in the region of the right kidney, extending into the thigh of the same side. Pressure over the right loin, directed inwards and upwards, immediately below the twelfth rib, was extremely painful. As long as he lay quietly on his right side, he was almost free from pain; on moving the trunk and on coughing, pain was increased. There was no blood in the urine. The pain soon passed away. Five days after the beginning of these symptoms he died. A large infarction was found in the right kidney; this protruded above the surface of the kidney, which was not true of other infarctions found at the same time.

As regards the diagnosis of hemorrhagic infarction of the kidney, it is evident, from the above statements, that it cannot generally be made during life. Whenever one of the fundamental diseases is present which give occasion to the formation of infarctions—if the symptoms arise which were present in Traube's case—one may suspect larger infarctions in the kidney.

Course. Prognosis.

Infarctions of the kidney are chronic in their course. A considerable time elapses between their first development and the formation of a cicatrix. The large number of older and more recent renal infarctions which are found on making post-mortem examinations—in cases where no change in the action of the kidneys was observed during life—teach us that, as such, they do not materially vitiate the prognosis. The latter depends on the character of the fundamental disease, and of other serious complications of the same, upon which we cannot here enter more specifically.

Therapeutics.

Treatment is powerless. At most, as in Traube's case, local abstractions of blood may be undertaken if there is excessive pain.

Necrosis of the Kidney.

LITERATURE.—The works given on page 543, and *Cohnheim*, Untersuchungen über die embolischen Prozesse. Berlin. 1872.

Etiology.

We have seen, when studying suppurative nephritis, that, as the result of dissecting inflammation and suppuration, individual portions of kidney tissue may be detached from their organic connections, and may be voided with the urine as dead parts (compare pages 556 and 557). Aside from this, however, necrosis of kidney tissue takes place in larger areas, when the renal arteries are obstructed, and when the conditions described on pages 605 and 606, which would admit of the occurrence of an infarction, do not exist. Those portions which are shut off from nourishment undergo necrobiosis. Necrosis without infarction is especially liable to follow obstruction of the trunk of the renal artery.

Pathology.

According to a careful observation of Bartels and Cohnheim (l. c., p. 76), the urine undergoes no morbid alteration in necrosis of *one* kidney, that has resulted form obstruction of one renal artery. The case was that of a boy eight years old, who suffered, among other things, from an *embolism of the entire left renal artery*, caused by extensive thrombus masses which existed in the left ventricle, and which were, in part, floated away into the peripheral arterial system. This kidney was increased in width, but particularly in thickness. The capsule, as well as the adipose tissue surrounding it, was swollen and juicy. The capsule was easily detached, its surface smooth and marbled. Quite irregular, washed-out, ill-defined red spots stood forth in contrast to a dull, yellowish gray, clay-colored ground. In consistency the organ was moderately firm. On section, the entire cortical substance appeared *clay-colored*, *juiceless*, *pale and dull*, *extremely opaque*, *as if dead*, while the pyramids were of a tolerably lively, bluish red color. In the midst of the cortical portion and in some of the pyramids, peculiar, deep bloody red spots were discernible, being irregularly distributed, and not sharply bounded. In these the Malpighian corpuscles could be recognized as red points, whereas they could not be distinguished with certainty in the clay-colored zone. The line of demarcation between the straight and convoluted divisions of the urinary tubules could be distinctly made out with the naked eye. All the larger arteries that were cut across by the section were filled with a firm, dark red plug, which could be traced backward, quite continuously, into the main trunk of the renal artery. With the exception of a piece one centimetre in length, at the beginning of the artery, this vessel was totally obstructed throughout its entire extent and in its larger branches. The larger veins contained only a little thin, fluid blood. The pelvis of the kidney was empty. Microscopically, the renal epithelium of the necrotic kidney was distinguished solely by its more decidedly granular character. The only blood-vessels in the organ which contained an abundance of blood were those situated in the dark red spots, and here, too, blood-corpuscles could be clearly demonstrated in the interstitial tissue of the kidney.

The swelling and thickening of the peri-capsular tissue might be regarded as the beginning of an inflammatory reaction of these parts, called forth by the irritation of the necrotic tissue in the kidneys.

Amyloid Degeneration of the Kidney. (Lardaceous or Waxy Kidney.)

Literature, Definition, and History.

In addition to the literature given on page 543 ·

Virchow, his Archiv VI. and VIII. Deutsche Klinik. 1859; also Cellularpathologie, Geschwülste. II. p. 616.—*Meckel*, Charité-Annalen. 4. Jahrgang. 2. Heft. reprint. —*Friedreich*, Virchow's Arch. XI. p. 393. XIII. p. 498.—*Beckmann*, ibidem, p. 94.—*Todd*, Clinical lectures on certain diseases of the urinary organs. 1857.—*Kekulé*, Verhandlungen des natur-histor. medic. Vereins in Heidelberg. 1858. p. 144.—*Traube*, Gesammelte Beiträge. u. s. w. 1871. II. 1. pp. 373 and 378. (These works date from the years 1858 and 1859); also *Traube*, Die Symptome der

Krankheiten des Respirat.- u. Circul.-Apparats. Lieferung 1. 1867.—*Neumann*, Deutsche Klinik. 1860. Nr. 37.—*Pleischl* und *Klob*, Wiener med. Wochenschrift. 1860.—*C. Schmidt*, Annalen der Chemie und Pharmacie. LX. 1859. p. 250.—*E. Wagner*, Archiv f. Heilkunde. 1861. p. 481.—*Grainger Stewart*, Edinb. med. Journ. Feb. 1861. August, 1864.—*Kühne* und *Rudneff*, Virchow's Archiv. XXXIII. 1865.—*H. Fischer*, Berlin. klin. Wochenschr. 1866. Nr. 27.—*Beer*, Eingeweide-syphilis. 1867.—*J. Wilks*, Guy's Hosp. Rep. 1865. p. 45.—*T. W. Parry*, the same. 1864. p. 315.—*Cohnheim*, Virchow's Arch. XXXIII. p. 155. and LIV. p. 271.—*Pilz*, Jahrbuch f. Kinderheilk. Neue Folge. III. reprint.—*Gerhardt*, Kinderkrankheiten. 1874. 3 Aufl.—Inaugural Dissertations by *Fehr* (Bern. 1866), *Muenzel* (Jena. 1865), *Tosca* und *Taesler* (Greifswald. 1867), *Wolff* (Berlin. 1869).—*E. Modrzejewski*, Archiv. f. experimentelle Pathologie. I. p. 426.—*Senator*, Virchow's Archiv. LX. p. 476.—*Johnson*, Brit. med. Journ. 1873.

Our views with regard to amyloid degeneration in general, and that of the kidneys in particular, were a long time in clearing up. The altered appearance of certain organs that had undergone this degeneration did not escape the keen observation of the older physicians. Antoine Portal, in 1813, in his Diseases of the Liver, p. 365, describes the liver of an old woman, who had various exostoses and tumors about the genitals, as "réduit en une substance pareille à du lard, soit pour la couleur, soit pour la consistance." Similar observations are found in Budd,[1] under the head of "Scrofulous Swellings and Hypertrophy of the Liver." At the same time none of these authors makes mention of simultaneous disease of the kidneys, although later experience has taught us that this is probably never absent in case of amyloid degeneration of the liver. Furthermore, these physicians had no clear conception of the nature and essence of these alterations in the liver. In 1842, Rokitansky first designated the lardaceous kidney (which had hitherto been regarded as simple Bright's disease) as a separate (eighth) form of Bright's disease. He depicted its gross anatomical condition, clearly set forth its essential characteristics, recognized the fact of its constituting but one member of a group formed by simultaneous affections of the liver and spleen, and noted the relation of these affections to certain definite cachexias. Rokitansky considered that, in this disease, the organs in question were infiltrated by a lardaceous, albuminous, translucent substance. For more than a decade these views of Rokitansky's remained unnoticed, until Virchow and Meckel almost simultaneously devoted themselves to investigating the substance thus deposited in these organs. The various etiological conditions of this affection were very fully set forth by Meckel. He regarded the essential point in the degenerative process to consist in the development of certain fats and lardaceous matters, which were more or less identical with cholesterine. He established the iodine and sulphuric acid reaction, distinguished "lard-red" and "lard-violet" as varieties of the same, and designated the disease as the lardaceous or cholesterine disease. Virchow pointed out the errors in Meckel's method of demonstration, taught that the reactions of cholesterine and lard were by no means identical, and attributed the changes in the organs concerned to the deposit of a peculiar substance, whose reactions suggested the cellulose group, and which, for this reason, he called amyloid matter. The labors of Friedreich, Kekulé, and Carl Schmidt, as well as the more recent ones of Kuehne and Rudneff have completely overthrown the former view, to the effect that the amyloid matter belonged to the group of carbohydrates. Modrzejewski has recently furnished still further proof of the albuminoid character of this substance, by showing that its ultimate analysis yields products identical with those of albuminoid substances. Aside from pathologico-anatomical and chemical questions, this affection of the kidneys has, of late, acquired increased interest from the fact that more definite results have been reached in the matter of its diagnosis, and, within certain limits, of its treatment as well. While Meckel, yielding to the demand for a nomenclature corresponding to the symptomatology, was satisfied with the name of Bright's disease, which at that time was still quite an imposing name, most important points bearing on this subject have been furnished by the labors especially of Todd, Traube, and Grainger Stewart, and it has been further enriched by an additional series of special investigations and reports of cases.

---

[1] On Diseases of the Liver, London, 1845.

With regard to the position which amyloid degeneration occupies in the system of pathology, it will be found that some authors, even at the present day, treat of it under the head of

Inflammatory Affections, and in connection with Bright's disease. The two processes, as we shall see hereafter, may complicate one another. On the whole, in view of its nature and its course, amyloid disease of the kidney is to be discussed under the head of the Degenerative Processes.

Etiology.

Virchow characterizes the etiology of amyloid degeneration briefly and strikingly when he says this disease is always the result of a cachexia. It is most frequently found associated with the syphilitic, scrofulous, or tubercular dyscrasia. But, aside from the fundamental diseases above designated, there is a series of other affections which are likewise, though not so frequently, followed by amyloid degeneration. Amyloid matter is sometimes developed when we cannot find the product of any such dyscrasia in the dead subject and when the history of the case has given no indication thereof.

In by far the greater number of instances the processes which give rise to amyloid degeneration are those characterized by tedious suppuration, especially resulting from chronic disease of the bones, or it may be of the skin and other soft parts.

So far as we know at present, amyloid degeneration is developed under the following conditions:

Diseases of the bones are particularly liable to develop it. In case of caries or necrosis, in scrofulous subjects, the kidneys are very liable to be attacked with amyloid disease. Rayer, even in his time, reported a large number of cases in which such affections as this were associated with albuminous nephritis—he was not acquainted with the division into amyloid degeneration. Gangrene and suppuration of the larger long bones especially predispose to this affection. It is also often developed in fungous inflammation of the larger joints in scrofulous subjects. Traumatic injuries, and especially gunshot wounds of the bones, with chronic suppuration, should likewise be mentioned here. In such cases but a few months from the rise of profuse suppuration suffice for the development of amyloid degeneration.

I would further call attention to some other causal conditions, viz., those diseases of the bone which follow rheumatic periostitis and *simple prolonged ulcerations of soft parts*—for instance, very chronic ulcers of the feet. The amyloid degeneration very often does not appear until the diseased process in the bone is healed. So far as ulcers of the feet are concerned, the observation is worthy of prominence that amyloid degeneration of internal organs is very apt to take place if these ulcers pursue a chronic atonic course; often, indeed, though by no means always, when, after lasting for years, they are preparing to heal. After Lindwurm, in 1862, had reported some observations to this effect, the matter was more carefully followed up in 1866 by H. Fischer. On observing a large number of chronic ulcers of the feet, he found that, without the development of any other dyscrasia, albuminuria was developed in seven per cent. of the cases, and in four per cent. of these it was occasioned by amyloid degeneration of the kidney. Since that time the number of observations bearing upon this point has very much increased. We know nothing of the more immediate conditions under which amyloid degeneration is developed.

Rokitansky gives *rachitis* as one of the causes of this condition. These cases have, at all events, thus far been rare, and it is not yet certain that, in such cases, the kidneys participate in the amyloid process.

In *constitutional syphilis*, on the other hand, amyloid degeneration of the kidneys occurs pretty often. Frequently, however, nothing but the remains of syphilis can be found, without a single active symptom. Occasionally our only information with regard to previous infection and its results is obtained from the history of the case; so that the amyloid changes are regarded as constituting part of the cachectic stage of the constitutional syphilis. It has been frequently asserted that the use of mercury also has a share in the development of this condition. All that can be said with certainty is that urine which contains mercury will now and then be found to be albuminous. But this is by no means always the case, as Kletzinsky thinks. Julius Müller[1] never found albumen in the urine when mercury was present. Kussmaul[1] believes that the albuminous character of the urine in mercurialism may be referred to a mercurial catarrh, and does not consider it as proved that mercury also causes amyloid degeneration. With regard to constitutional syphilis, one observation which has been made repeatedly is of interest and of practical significance, viz., that it by no means only attacks persons who are badly nourished, but also those with a well-developed fatty layer and powerful muscles. Rokitansky has seen amyloid degeneration in congenital syphilis.

---

[1] Arch. f. Pharmacie, CXCIV. p. 9.

Chronic pulmonary phthisis is not infrequently associated with amyloid degeneration. E. Wagner found it in seven per cent. of his tuberculous patients. Even Meckel refers to the fact, which may often be verified, that, during the development and progress of amyloid degeneration, no progress is to be observed in the development of the pulmonary disease. Further investigations have even taught that more frequently a shrinking of the diseased portions of lung takes place. In some few cases the primary pulmonary affection is so slight that it escapes notice during life. It would be a matter of practical interest to inquire whether and how often phthisical patients who were the victims of amyloid degeneration were at the same time suffering from syphilis, or had previously suffered therefrom. Amyloid degeneration is sometimes developed after long continued *intermittent fever*, which has led to a breaking down of the constitution. Oedmannson[2] declares that this is often observed in Sweden after inveterate intermittent fever.

In some few instances amyloid degeneration is also observed in connection with *cancer*. E. Wagner met with it three times in 109 cases, though in but one of these were the kidneys involved. According to the few observations thus far on record, it would appear that carcinoma of the uterus is especially likely to be associated with amyloid disease. Blau[3] found amyloid degeneration of the kidneys four times among ninety-three cases of carcinoma of the uterus in the Berlin Pathological Institute. In one case in which Waldeyer made the autopsy, and in which there was medullary cancer of the right kidney, a portion of it, which was not the seat of cancer, as well as the other kidney, which was not cancerous, was in a state of advanced diffuse nephritis with amyloid degeneration.

---

[1] Constit. Mercurialismus. 1861. p 326.

[2] Schmidt's Jahrbb. 117. p. 156.

[3] Inaugural-Dissertation. Berlin. 1873.

Aside from the causes already indicated, various diseases, and especially *suppurative processes in various organs*, are associated with amyloid degeneration. Dickinson regards the occurrence of chronic suppuration as the most important etiological condition. Among sixty cases of amyloid disease, fifty-two were associated with suppuration. It is as impossible as it would be useless here to mention all the localizations that have been observed. As examples thereof we may mention long-continued fistulous ulcers in empyema, bronchiectatic cavities, and chronic bronchitis, generally accompanied with profuse suppuration, large ulcerated surfaces within the intestines, and lupus exulcerans. In calculous and other forms of pyelitis and nephropyelitis of *one* kidney, the other sometimes undergoes the amyloid change. Among the other processes of disease liable to be followed by amyloid degeneration we may here mention chronic interstitial nephritis. Virchow[1] has also found it in the cachexia combined with nephritis after scarlet fever. Johnson considers the long-continued albuminuria of chronic Bright's disease as one of the main causes of amyloid degeneration of the kidneys, of chronic peritonitis, of chronic muscular and articular rheumatism. A case is reported from Mosler's Clinic[2] in which no other ground for the occurrence of amyloid disease of the abdominal organs could be discovered than the fact that the patient had suffered from twenty-one attacks of pneumonia in fifteen years. I once found amyloid degeneration of the abdominal organs together with a large echinococcus cyst of the liver (not suppurated) in a man twenty-seven years old. Finally, there is a series of cases in which neither the history nor the post-mortem examination indicates any primary affection which might be regarded as standing in an etiological relation to this disease. Wilks designates such cases as "simple lardaceous disease." It is reserved for the future to discover the genetic relations here existing.

---

[1] *Virchow's* Archiv. VI. p. 271.

[2] *Posca's* Dissertation.

The attempt has often been made to determine the statistics of the etiological relations of amyloid disease by the analysis of a larger or smaller number of cases. Fehr has very carefully compiled such a collection, reaching to the year 1866. So far as I have been able to obtain it, I have compared therewith the material furnished up to the most recent date. The result remains the same. Constitutional syphilis, phthisis of the lungs and the intestines, and caries, generally in scrofulous subjects, occupy the first rank. The importance of these etiological factors, as bearing on the diagnosis, will be more fully discussed hereafter.

So far as *age* and *sex* are concerned, it appears that the majority of cases occur during middle life, when the primary affections, too, are most frequently observed. No age enjoys complete immunity. Men are attacked oftener than women.

Pathology.

Pathological Anatomy.

Kidneys that have undergone amyloid degeneration do not by any means always present the same anatomical picture. As long as the degeneration is confined to certain portions of the circulatory apparatus—as long, for instance, as the vascular loops of a portion only of the Malpighian corpuscles are affected—the kidney may present an apparently normal appearance to the unaided eye ; but as soon as the amyloid change advances, the diseased kidney increases in volume. This increase in volume may be very considerable. It is quite notably confined to the cortex, which is sharply distinguished by its light yellow color, like that of butter, from the pyramids, which are usually red. The amount of blood contained in the cortical portion is greatly diminished ; the blood which escapes from the vessels divided is always light colored and of a thin fluid consistency. According to Johnson, the blood in amyloid degeneration of the kidneys is poor in hæmoglobin and albumen, but rich in urea. Aside from this, the cortex presents a homogeneous appearance, the enlarged Malpighian tufts appearing, as Meckel expresses it, like sparkling dew-drops. It is only when the pyramids participate to a higher degree in the degeneration that they also grow pale and shining, and the kidney assumes a uniform, consistent appearance. The capsule is easily separable without any loss of substance. The pyramids are very seldom seen to degenerate alone, and then it would appear that in all the cases hitherto observed, the amyloid infiltration is confined to the thickened walls of the canals.

The diagnosis of amyloid degeneration on the dead subject is pre-eminently chemical, rendered possible by the peculiar reaction of amyloid matter. For the coarser demonstration thereof, it is sufficient to pour the solution of iodine upon the cut surface carefully cleansed from blood. In advanced cases this is easy, on account of the great lack of blood in the organ. Biliary coloring matter interferes with the amyloid reaction. In case of but slight amyloid degeneration, the negative result obtained from this test, with the naked eye, is not conclusive. If the test succeeds, those portions of tissue which have undergone the amyloid change show a mahogany or ruby-red color. If sulphuric acid is afterwards poured over them, these parts assume sometimes a brown, sometimes a more violet, and even a blue color. As the amyloid degeneration involves the finer and the finest arteries and capillaries, if any large proportion of these are diseased, one obtains a splendid view of the distribution of the circulatory apparatus. Amyloid degeneration of the veins is very rare. It has been described by Friedreich. For more accurate study, microscopic examination of the organ is indispensable, this being undertaken partly in its fresh state, on fine sections made with a double knife, and partly in the hardened state. The fine sections, which, if taken from the fresh preparations, should first be washed in distilled water, may be laid either into a weak solution of iodine and iodide of potassium, or of diluted tincture of iodine, or into a watery solution of iodine, and afterward, when the preparation is placed on a slide and under a glass cover, a drop of sulphuric acid is allowed to flow to it slowly, so as to cause no disturbance. After some time the color produced by the iodine will change either to a deep brownish red, or a dirty violet or blue. In some cases the iodine color does not change on the addition of sulphuric acid. In the central portion of the glomerules a bluish green color is developed, sooner or later, from the violet. Sometimes the iodine alone produces a violet color, which in these cases, on the addition of sulphuric acid, is transformed into the most beautiful clear blue. Solutions of iodine and iodide of zinc (Munk), of chloride of zinc or chloride of lime (Bernhardt), have also been recommended as tests for amyloid matter. At the same time, and it appears to me justly so, the iodine solution has remained practically the one most commonly in use.

So far as *the distribution of amyloid matter in the circulatory apparatus of the kidney* is concerned, in by far the greater number of cases the Malpighian bodies are first attacked. (This fact, so far as I can see, is denied by no one but Johnson.) The next in order are the arteriae rectae, as well as the vasa afferentia, the vasa efferentia being but seldom involved. The various preparations from the same kidney, however, do not by any means always show alterations to the same extent or of the same intensity. The capillary system surrounding the urinary tubules does not become involved unless the amyloid degeneration has reached a high grade. In some isolated instances the renal artery itself shows amyloid degeneration, while the arterial branches lying between the straight tubules are not yet attacked.[1] Occasionally the urinary tubules are involved in the degeneration, which in part attacks their investing membrane and in part their epithelium—far more frequently, however, the former than the latter. Now and then all the vessels and all the tunics proper of the tubules have been found involved. Amyloid degeneration of the ephithelial cells is an extremely rare occurrence, and one which is entirely denied by some observers. These cells may be transformed into glassy, clod-like masses, which, like the tunics proper, give the same reaction as the blood-vessels, and which fill the entire calibre of the urinary tubules. Sometimes, also, more extensive masses of amyloid matter are found in the kidneys. Beckmann found in the renal pyramids of an old suicide snowy white spots and stripes, very distinctly recognizable by the naked eye, which were formed of perfectly pure amyloid masses. In the cortical portion the Malpighian bodies were but slightly altered.

---

[1] *Demme*, Schweiz. Zeitschr. f. Heilkunde. I. p. 117.

What, now, does the microscopic examination of vessels that have undergone amyloid degeneration reveal? There are peculiar alterations, which can first be distinguished in the vascular coils of the Malpighian bodies. The capsule appears thickened, but the nuclei of its epithelial cells are generally distinctly recognizable. The Malpighian bodies appear enlarged, and the capillary loops of which they are composed are thickened, of a dull lustre, translucent, homogeneous, and seem to be transformed into a structureless mass. Quite an analogous sclerotic condition is also sometimes observed, in which the vascular coils of the Malpighian bodies are transformed into a dense mass, but where the iodine and sulphuric acid test gives no result.

Quite similar changes to those presented by the capillary walls in amyloid disease may be found in the walls of arteries of larger calibre that are thus attacked. Here it can often be shown how the process, beginning in the inner tunic of the vessel, extends from there to the muscular layer, and in extreme cases to the outer coat. Sometimes the muscular coat alone shows the amyloid reaction; its constituent elements seem to run together, and are no longer distinguishable, the muscular coat of the vessels appearing thickened and quite transparent. This increase in volume of the amyloid tissues, if it assumes larger proportions in an organ, almost always causes an enlargement of the same.

It has been urged, especially by Virchow, that *it is not practicable to make a complete injection of the vascular apparatus* of such kidneys on account of the narrowing of the finer vessels, caused by the infiltration of their walls with amyloid matter. Even the finer materials which we use for injections are far too coarse to pass through the narrowed vessels. Muenzel[1] succeeded in the case of two kidneys, one of which was highly degenerated, in injecting the Malpighian bodies, and beyond these the second capillary network of the cortical and the medullary portions, thus demonstrating that the diameter of the vessels, in kidneys which are the seat of amyloid disease, may range within the same limits as those ordinarily presented by the normal kidney after injection. There must, therefore, be kidneys the seat of advanced amyloid degeneration whose vascular system remains perfectly permeable to the blood-stream.

---

[1] Jenenser Dissert. 1865.

With regard to the conduct of the epithelium of the kidneys in amyloid degeneration, I have already said that in extremely rare instances it may be involved in this process. In another series of cases it remains normal. In still other cases, however, changes take place in the epithelium which fall chiefly in the domain of cloudy swelling, terminating in fatty degeneration and subsequent atrophy. Muenzel also describes a proliferation of the same.

The alterations in the epithelium have generally been regarded as a remote result of the degeneration of the blood-vessels, it being considered that the nutrition of the epithelial cells suffered on account of the insufficient supply of blood due to the great narrowing of the vessels. At the same time this method of explanation does not cover, or only partly covers, all those cases in which the arterial avenues are not at all, or but little, diminished in calibre. On the whole, it seems very probable that these degenerative processes in the epithelium, and the amyloid degeneration itself, are manifestations of the same fundamental disease, and depend on the blood-changes caused by such disease. At all events, these epithelial changes represent *degenerative processes*, whether as the result of local or general disturbances of nutrition, and not merely "parenchymatous inflammatory processes."

Many of the urinary tubules are filled with albuminous casts, some of which fill their calibre entirely, while others leave a considerable interspace between the cast and the epithelium. These casts are sometimes quite homogeneous, sometimes finely granular, and occasionally of a light yellow color. They have never given me the amyloid reaction. Even at the present day the strife is not yet settled as to whether the occurrence of gelatinous casts in the kidneys is caused by the coagulation of some albuminoid substance transuded through the capillary walls in a soluble condition—therefore blood-fibrin or exudation-fibrin—or whether it is to be regarded as the result of a change in nutrition of the glandular epithelium. I believe that the casts may arise in both ways. This much is certain, that casts may be formed in the urinary tubules while the epithelium is quite normal (compare, for instance, the observations of Muenzel). The fact that very few casts are voided with the urine of patients suffering from amyloid degeneration of the kidneys, must not be taken as evidence that but few casts are found in the urinary tubules. For it is well known that thick casts developed in the convoluted tubes cannot reach the urine, as they would certainly have to pass the much narrower loops.

With regard to pathological changes in the interstitial tissue in amyloid degeneration of the kidneys, it would appear that these only attain a high grade when the degeneration is the result of syphilis. In that case there may even be a shrivelling of the organ, which is extremely rare in the amyloid kidney, developed in consequence of pulmonary phthisis, suppuration of bones, etc. A. Beer considers diffuse, cellular, interstitial hypertrophy, extending over the entire kidney with lardaceous degeneration of the vessels and manifold stages of retrograde metamorphosis of the newly-formed masses as well as peculiar parenchymatous alterations, especially little fatty accumulations, as directly characteristic of syphilis of the kidney. These cases represent the worst and clinically the most important form of diffuse syphilis of the kidneys, which, according to Beer, very seldom occurs as the result of syphilis without lardaceous degeneration of the vessels. In these cases of syphilitic amyloid kidney there is usually also amyloid degeneration of the other abdominal organs. Whether the amyloid degeneration is here the primary process which, as Munck thought, favors the development of interstitial nephritis as the result of the escape of blood through ruptured vessels, which would act as an exciter of inflammation, or whether the reverse of all this is the case, or, finally, whether both processes arise simultaneously; these are, in my opinion, still open questions which cannot at present be determined on the basis of positive demonstration. Where, in cases of lardaceous kidney, the highest degree of atrophy exists

—shrivelled Malpighian bodies with thickened capsules and very small kidneys—there, according to Beer's idea, which certainly has the probabilities in its favor, the kidney was already shrivelled when amyloid degeneration of the vessels set in, because amyloid matter is incapable of any retrograde metamorphosis. In case of primary amyloid degeneration, then, shrinking of the kidney would only be able to occur up to a certain point. Aside from this, cases also arise (Klebs) in which very extensive interstitial cell-infiltration occurs in the kidneys, together with slight amyloid degeneration of these organs, in which no amyloid change is found in any other organs, and in which all other known causes of this degeneration may be wanting.

Amyloid degeneration of the kidneys is generally bilateral, commonly, though not always, being of equal intensity on the two sides. It sometimes happens that only *one kidney* degenerates in this manner when, for instance, the other is altogether lacking, or when it is the seat of extensive suppurative processes or of cancerous disease. Ruehle describes an interesting case belonging in this category. The left kidney was greatly enlarged and infiltrated with amyloid matter (200 grms. [℥ vi. ʒ iij.] in weight); the right kidney was strikingly diminished in size (60 grms. [ʒ xv.] in weight), completely granular, but not amyloid. At the same time there was amyloid disease of the spleen and hypertrophy of the left side of the heart.[1]

In case of amyloid degeneration of the kidney, we often find the same process taking place in other abdominal organs. The order of consecution in which these organs are diseased is not uniform even in the same fundamental disease—much less in different diseases. Among 80 cases of amyloid degeneration, Carl Hoffmann[2] found the spleen attacked 74 times, the kidney 67, portions of the intestine 52, and the liver 50 times.

With regard to the nature of amyloid matter, I have already stated above (page 611) that, according to the present state of our knowledge, this substance seems to belong to the class of albuminoid substances. It has been obtained in greatest purity by Kuehne and Rudneff, in the form of a snowy white substance, which reacted with special beauty to the iodine and sulphuric acid test. Although amyloid matter in many respects resembles albumen, especially coagulated albumen, yet it has also many peculiarities differing from those of albuminoid bodies. The similarity of the two substances lies in the following points, viz.: a great analogy in their chemical composition; both give the xanthoprotein reaction and respond to Millon's test, and, as Modrzejewsky has recently demonstrated, if they are boiled in a dilute acid, they conform to leucin and tyrosin in the products into which they subdivide. On the other hand, aside from the iodine reaction which is peculiar to it, amyloid matter is to be distinguished by its great power of resistance to many solvents, and especially by its utter insolubility when digested with dilute gastric juice. Furthermore, amyloid matter does not putrefy, even in the course of months, and offers the most obstinate resistance to the inroads of suppuration. With regard to the causes which give rise to the development of amyloid matter in the human body, as well as to its being deposited in certain tissues of the lower animals, we do not as yet know anything positive. Virchow considers it as tolerably probable that in amyloid degeneration there is a gradual permeating of the parts with a substance which is brought to them from without. He is inclined to the opinion that amyloid matter, or a substance one step below it, is to be found in the fluids, to which it is brought by some local disease. Opposed to this is another hypothesis, to the effect that amyloid matter is formed on the spot, out of albuminates there deposited. Friedreich assumed outright that it resulted from the gradual transformation of fibrin. Dickinson[1] accounts for the origin of amyloid degeneration solely by the loss of albumen and of alkaline carbonates which the blood undergoes during chronic suppuration, seeing that pus contains more of these substances than the blood itself. He says that amyloid matter, which is essentially fibrinous in its character, instead of assuming a yellow color, like fibrin and the normal tissues, takes a characteristic reddish brown color, in consequence of the absence of the alkalies, which is immediately lost if some carbonate of potassium is added to it. Dickinson claims that, by the withdrawal of the alkalies combined with the albuminates, amyloid degeneration may be artificially produced.

---

[1] Greifswalder Beiträge.

[2] Dissert. Inaugural. Berlin. 1868.

---

[1] Essay read before the Med. and Chirurg. Society of London, February, 1867.

Symptomatology.

The symptoms which are presented by patients with amyloid degeneration of the kidneys vary according to the fundamental disease which has occasioned the renal affection; according to whether chronic disease of the bones, pulmonary phthisis, or some of the manifold manifestations of constitutional syphilis are present; according to whether the kidneys alone are involved in the degeneration, or the liver and other organs are also affected. We shall here only notice the symptoms dependent on the renal affection. Symptoms dependent on diseases of other organs will only be referred to so far as they have a bearing on the diagnosis, or materially influence the course of the affection.

Persons affected with amyloid degeneration of the kidneys are generally the victims of some wasting disease. They are emaciated, pale, anæmic, complain of increasing weakness and loss of flesh, and, even if the fundamental disease should not have such an effect, they are always tired and unfit for work. This is the rule, but it also has its exceptions, and one would often be misled if he should exclude the possibility of amyloid degeneration of the kidneys in well nourished, muscular individuals. Great paleness of the skin, however, would hardly ever fail to be found. Well-nourished individuals with amyloid kidneys are especially found among the victims of constitutional syphilis. It appears to be beyond a doubt that amyloid disease of the kidneys may follow comparatively early on primary syphilitic infection, at a time when the patients are still well preserved, and furnished with a good padding of fat and a well developed muscular system.

The symptoms occasioned by the amyloid affection of the kidney, as such, are at first almost entirely dependent on *changes in the urine and in the secretion of urine.* So far as the quantity of the urine is concerned, we, unfortunately, do not find that unanimity on this subject, amongst authors, which we might desire. English authors regard an increase in the quantity of the urine as the earliest symptom. According to Stewart,[1] patients begin by passing copious quantities of urine and drinking much. In some cases patients do not notice the increased amount of the urine, and are only troubled because they are obliged to urinate repeatedly during the night. Stewart's assertion that the quantity of urine is increased is opposed by his own figures, however, according to which the quantity varies greatly, being from 1,080 to 6,000 c.c. in the twenty-four hours, for in the first case one would have to assume a diminution rather than an increase. Johnson expresses himself similarly to Stewart, viz., that the first symptom of amyloid degeneration of the kidneys is the appearance of an abundant secretion of pale urine of low specific gravity. Taesler, too, found the quantity of urine increased in the first and middle stages of amyloid disease of the kidney. According to the observations of Traube, the volume of the urine is at first abnormally large, or just about normal, in those cases particularly where the amyloid disease is developed in the course of chronic pulmonary tuberculosis. From these various statements thus much may be deduced, *that an increase in the quantity of urine at first does not constitute a characteristic symptom, but that where it is observed it is not to be underestimated.*

The *reaction* of the urine is feebly acid, the color light yellow. The specific gravity is generally low, though in some cases normal, varying from 1006 to 1016 and 1017. The urine is usually *rich in albumen.* It is clear, sometimes shows no sediment, or, after standing for a long time, may deposit a slight sediment of a whitish color, which can hardly be collected on a filter, and which consists of *a few pale hyaline casts, some lymph-corpuscles, and a few partly fatty epithelial cells.* In by far the majority of cases neither the casts nor the cells show the amyloid reaction. Only a few observers—among them Stewart in particular—claim sometimes to have seen the amyloid reaction in the renal casts. The same has been observed quite exceptionally in the epithelial cells. The quantity of urea is generally diminished, the uric acid likewise; sometimes a complete lack of the latter has been observed; sometimes a diminution in the excretion of phosphates and chlorides has been observed.[1] Senator designates the urine in amyloid degeneration of the kidneys as a non-inflammatory transudation due to stasis, which has been pressed through the vascular coils and is thinned with urine. According to him, in addition to serum-albumen, it also contains (para-) globulin in more distinct and larger quantities than does other albuminous urine, and perhaps also alkaline albuminates. Its *feebly* acid reaction, too, probably depends upon an admixture of the transudation with the urine (Senator).

---

[1] Edinb. med. Journ. August. 1864.

Sometimes the urine retains the above-mentioned light, clear quality until death. In other cases, on the contrary, it becomes scanty, while containing the same quantity of albumen. It is then red, of a high specific gravity, and has a tendency to throw down the urates. According to the observations of Traube, this state of things arises when a febrile affection is developed in the course of the disease, or when, in addition to the amyloid degeneration of the kidneys, conditions exist which lead to great congestion in the venous system.

The relations of the urine, as depicted above, are subject to many variations. In the first place, in the same patient the albumen may temporarily disappear from the urine, without being accompanied by any improvement. In fact, there are cases described in literature where, in the course of the entire disease, no albumen was observed in the urine. Fluctuations in the amount of albumen are very frequent. Furthermore, the fibrinous casts, which in the majority of cases are but few in number, may be abundant, and sometimes they are covered with epithelial cells containing granular matter. In such cases, where there is a considerable deposit of organized tissue-elements, we may be sure that something more than the degeneration of the vessels of the Malpighian bodies alone has taken place, that the degeneration has extended to other blood-vessels or to the epithelial cells, or that there is a complication with some other processes, such as those of congestion or of inflammation. In some cases *no casts* are to be found. In one instance Virchow observed an extremely large proportion of pigment matter in the urine. Here the cortical portion of the supra-renal capsule was found to be the seat of extreme amyloid degeneration. As a general rule, notwithstanding the presence of an especial abundance of serum-albumen, there is no blood in the urine. Blood-corpuscles are but very rarely found, and then usually after a long search.

---

[1] *Taesler* (l. c.) and *Dickinson*, Pathology, etc., of Albuminuria. 1868.

Amyloid degeneration of the kidney is a non-febrile process. Taylor declares that the temperature is lowered in this affection. This statement awaits confirmation. Elevations of temperature are encountered when caused either by the fundamental disease, or by some complicating febrile process.

In the early stages of amyloid degeneration of the abdominal organs there is absolutely no *dropsy*. Sometimes none is developed even during the further course of the affection. Todd narrates a well-marked case, which occurred in the person of a medical man, where there was no sign of dropsy after the disease had lasted two years. But where, in amyloid degeneration of the abdominal organs, the kidneys are also involved, dropsy is more frequently present than absent. But the period of its occurrence is very variable. Sometimes, developing itself rapidly, it only appears towards the end of life as a severe terminal symptom. In other cases œdema about the ankles may exist for months, only to increase during the last stages. Sometimes such œdema about the malleoli, appearing only at evening, after the patient has gone through with the labors of the day, and vanishing during rest in bed, is the first sign that attracts the notice of the patient. Dropsy may remain confined to the lower extremities in the later stages too, but sometimes, during the further course of the disease, effusion takes place into the cavity of the chest or abdomen. According to Murchison,[1] the latter only occurs when enlarged lymphatic glands compress the trunk of the vena porta as it enters the liver.

Digestion is sometimes in nowise disturbed, notwithstanding amyloid degeneration of the kidneys. I have repeatedly observed, in hospitals, that patients, particularly those with constitutional syphilis, who had the most unmistakable signs of amyloid kidneys, enjoyed the best of appetites. In the later stages, however, digestion is, sooner or later, more or less disturbed, especially if amyloid degeneration is also developed in the vessels of the mucous membrane of the alimentary canal. The same thing may occur in the earlier stages, too, if digestion is disturbed from the outset by the fundamental disease; or if complications in the digestive organs are developed during the course of the disease, such as extensive ulceration of the intestines; or if, as the result of the primary process, signs of impeded circulation appear in the region tributary to the inferior vena cava, with consequent catarrh in different portions of the digestive tract. Under these circumstances the appetite is lost, there is a disgust for food, nausea, and not seldom even obstinate vomiting. As a sequel to chronic intestinal catarrh, extensive ulceration of the intestines, and amyloid degeneration of the vessels of the intestinal mucous membrane, profuse, almost uncontrollable diarrhœa is developed, which hastens the fatal termination.

---

[1] Dis. of the liver. London. 1868. p. 452.

Symptoms referable to the *heart* and *nervous system* are just as rare, following amyloid degeneration, as they are frequent in diffuse nephritis. Hypertrophy of the left ventricle occurs but seldom. If present, it is generally in cases in which the general conditions of nutrition have not yet suffered very much, in which the degeneration of the kidneys is already quite extensive, and especially in which, in addition to amyloid degeneration, contraction of the kidney either exists or is being developed. In a word, it is the more likely to be found the more obstacles exist in the aortic system, which must be overcome by the left ventricle. The circumstances peculiarly favorable to the occurrence of hypertrophy of the left ventricle present themselves under two contingencies: viz., 1st, when amyloid degeneration of the kidneys has been developed in addition to constitutional syphilis in a well-nourished individual; 2d, when amyloid degeneration has been developed in connection with pulmonary phthisis, the latter disease receding, and the conditions of nutrition consequently improving. In both cases the amyloid degeneration must of course be sufficiently extended to induce the requisite obstruction in the arterial system. The fact that these contingencies are very seldom met with in extensive degeneration of the kidneys, which usually very soon leads to cachexia, explains why hypertrophy of the left ventricle is not only exceedingly rare, but also hardly ever attains the same grade as in simple shrinking of the kidney, the result of diffuse nephritis. Hypertrophy is developed slowly and gradually, and manifests itself by the symptoms described under the head of Hypertrophy of the Left Ventricle.[1]

Symptoms of uræmic poisoning, like those of hypertrophy of the left ventricle, are also extremely rare in amyloid degeneration of the kidney. They have thus far been observed in those cases where there was, at the same time, hypertrophy of the left ventricle, although they were by no means constant even there. The retina is hardly ever diseased in amyloid degeneration of the kidney.

Complications and Sequelæ.

The most frequent complications are amyloid degeneration of the liver, spleen, and the mucous membrane of the digestive tract. Aside from this, amyloid degeneration of the kidneys, like other renal diseases which are capable of diminishing the excretion of the essential solid constituents of the urine, favors the occurrence of inflammatory processes. First on the list stand inflammations of serous membranes, especially of the peritoneum, which may give rise to inflammatory effusions into the cavity of the abdomen. Pneumonia and pleurisy are also not rare. Pericarditis, on the contrary, is but seldom observed. Extensive suppuration of cellular tissue is now and then given as a complication of the amyloid processes. In some cases, too, the so-called hemorrhagic diathesis is developed, now and then giving rise to obstinate hemorrhages from the nose and from other organs, which quickly debilitate the patients. Thrombosis of the femoral veins not infrequently occurs in consequence of a high degree of marasmus, causing œdema of the thigh. Sometimes other disorders of the urinary organs are developed in addition to amyloid degeneration of the kidneys, for instance, pyelitis, which causes the appearance of abundant quantities of pus in the urine, a condition never met with in simple amyloid degeneration, and which may greatly embarrass the diagnosis. Under these circumstances the urine generally contains a far greater amount of albumen than would be contained in the pus-serum present.

---

[1] See Vol. VI. of this Cyclopædia.

Diagnosis.

In order properly to recognize amyloid degeneration of the kidneys, it is of the utmost importance that due regard be paid to the etiological conditions above set forth. If albuminuria arises as a sequel of constitutional syphilis, chronic pulmonary tuberculosis, tedious suppurative processes, especially suppuration of bone-tissue, we have already reason to fear amyloid degeneration of the kidneys. Even an increase in the amount of the urinary secretion during the twenty-four hours, without the presence of albumen, is worthy of attention. Suspicion is increased to the highest degree of probability if manifestations arise on the part of the spleen, the liver, and the digestive canal, which might indicate similar disease of these organs. The condition of the urine is, furthermore, of the greatest consequence to the diagnosis. The conditions which it usually presents have been indicated on p. 625.

The urine is ordinarily clear, light yellow, transparent, feebly acid, very poor in morphological ingredients, and very often of low specific gravity. All this, it is true, may also be the case in the urine of contracted kidneys; here, however, the specific gravity is generally *much lower* than in amyloid degeneration, in which it probably never falls below 1006. For in contracted kidneys the urine usually contains far less albumen and urea. We have seen above that, according to the observations of Traube, heavy, red urine, rich in albumen, whose color depends on the presence of an abnormal quantity of urinary coloring matter, may, under some circumstances, be encountered in amyloid degeneration. In such cases, Traube decides in favor of amyloid degeneration, if the patient has for some time suffered from dropsy, which is evidently not caused by abnormal pressure in the venous system, developed as the result of one of the above-named etiological conditions, and if a *considerable* tumor of the spleen can be demonstrated which cannot be regarded as the result of intermittent fever. In contraction of the kidney the urine does not assume a red color, even when febrile complications occur, or stasis takes place in some portion of the venous system. How far the statements recently made by Senator with regard to the urine in amyloid degeneration of the kidney (see p. 626) will be confirmed, remains to be proved by further observations.

The absence of hypertrophy of the left ventricle, other things being equal, argues far more for amyloid degeneration than for contracted kidney. By means of the considerations given above, a diagnosis can, in most cases, be made with considerable probability of its being correct. But in case of the failure of some of these points, and especially of the etiological evidence, the diagnosis may present insurmountable difficulties.

Duration, Termination, and Prognosis.

It is difficult to arrive at any general conclusion with regard to the duration of kidney affections, because it is only in exceptional cases that the first beginning of the disease can be established with anything like accuracy. Stewart dates the beginning of the disease from the occurrence of polyuria. But no great weight can be attached to this, because the symptom is inconstant. There is, however, an abundance of clinical observations going to prove that the disease may exist during a series of years, and that it may be comparatively well borne, if the disease lying at the foundation thereof does not assume a pernicious course, and especially if amyloid degeneration of the digestive organs does not supervene. This is particularly true among patients of the better class, who can surround themselves with every comfort, and thus counteract the effects of the disease, and particularly make up for the daily loss of albumen. The prognosis, however, depends very much upon the character of the primary affection which occasions the amyloid degeneration. It is found to be comparatively the most favorable in syphilis, because this disease offers the best points of assault to therapeutics. Even in phthisis, in spite of complication with amyloid degeneration, the course of the disease may be very chronic, provided that the pulmonary trouble comes to a standstill, which happens not seldom. The literature of the subject even contains some cases of the cure of amyloid degeneration. Gerhardt considers the prognosis, especially during the period of childhood, as far more favorable than it is generally thought to be. When the fundamental disease is curable, the amyloid degeneration, according to the statements of this observer, can pretty certainly be removed. Notwithstanding all this, the disease is a very grave one, which generally, sooner or later, leads to death. The temporary disappearance of albumen from the urine does not justify a more favorable prognosis, unless it is accompanied by an improvement in the general manifestations, because experience has taught that the albumen may disappear for a while, or even be entirely wanting, while the disease still advances in its dread career. Death rarely supervenes through simple amyloid degeneration of the kidneys, but generally as the result of complicating processes, whether in the form of extension of the amyloid degeneration to several organs, or of secondary inflammatory processes. Sometimes death occurs from simple marasmus.

Therapeutics.

The first task imposed upon therapeutics is prophylactic in character. In case of the presence of any recognized fundamental disease, we should labor to prevent the development of amyloid degeneration. Unfortunately, we are without the means of accomplishing this task. We only know that the longer these processes last the more the danger of amyloid degeneration increases. To cure these processes as speedily as possible, therefore, presents itself as our first and most important duty. Experience has furthermore taught that amyloid degeneration, as the result of syphilis, may arise tolerably early. The practical importance of making early and frequent examinations of the urine, in persons suffering from constitutional syphilis, is therefore apparent. If treatment is to be of any avail, it must begin early. Then, under appropriate treatment, one may sometimes see moderate albuminuria with œdema of the lower extremities disappear, though, unfortunately, this only takes place in isolated cases. The treatment most to be recommended consists in the use of preparations of iodine, small doses of iodide of potassium, iodide of iron, etc. Grainger Stewart[1] saw a case of amyloid degeneration of the kidneys, caused by syphilis, almost (?) entirely cured under the use of the syrup of the iodide of iron. A material diminution in the tumors of the liver and spleen, which were referable to the same etiological condition, was also observed under this treatment. In one case (observed by Ruehle, *Greifswalder Beiträge*), in a syphilitic woman, the use of alkalies effected a very material diminution in the size of the liver and spleen, and a very decided lessening of the albuminuria. Although, unfortunately, these methods of treatment very often leave us in the lurch, still a careful use of them should always be made in analogous cases. Even when amyloid degeneration associates itself with pulmonary phthisis, therapeutic interference is often of decided benefit. Here cod-liver oil commends itself as an admirable palliative. Even if the renal disease remains unchanged, the losses induced by the disease are made good by this means and by appropriate nourishment. Indeed, the patient sometimes increases in weight, and his pale cheeks grow red. With the increasing density of the blood the dropsy, too, often disappears, a gain which is of the greatest importance. In other cases dropsy has been seen to disappear, under heavy sweating, while the patient remained quietly in bed and took no medicine whatever. In other patients powerful diuretic remedies not only increase the secretion of urine, but also temporarily increase the amount of urea. Among the means that have proved availing in this respect is a solution of borotartrate of potassium (from one-half to one ounce in six fluidounces of water). The decoction of cinchona has also developed a diuretic action in some cases. If there is no diarrhœa, the decoction of cinchona may be combined with bitartrate of potassa. If diarrhœa exists, the watery extract of nux vomica may be used instead. In order to augment the diuretic action, rubbing with Stokes's liniment may be practised from time to time. Moderate diuretic action has also sometimes resulted from the use of lactate of iron (two grains to the dose). These are about the methods whose employment has proved of some avail, even though it be only in isolated cases. Unfortunately, in the majority of instances, the process strides on, unchecked, in spite of all therapeutic endeavors. We must then content ourselves with maintaining the powers of the patient as long as possible, and preventing cachexia by means of bland, nourishing diet, and, so far as possible, with mitigating the sufferings of the patient—which are often very great—by judicious symptomatic treatment.

---

[1] Med. Times, June 7, 1873.

Tumors of the Kidney, of the Pelvis of the Kidney, and of the Perinephritic Tissue.

LITERATURE.—The works alluded to on page 543, and *Virchow's* Geschwülste.

Beginning with the consideration of *connective tissue neoplasms*, we will first deal with *fibromata* (nephritis interstitialis tuberosa of Virchow). These have no clinical importance. They are generally found in the form of little nodules, from the size of a lentil to that of a cherry-stone. They usually consist of a very dense fibrous tissue, within which run a number of atrophied uriniferous tubules. They are sometimes met with in connection with diffuse interstitial nephritis, sometimes without it.

Lipomata and *Myxomata* are likewise of no clinical significance. The first are encountered as *sub-capsular* lipomata. They consist of a somewhat lobulated fatty tissue, and must not be confounded with multiple supra-renal capsules to be found at the same spot and of a sulphur-yellow color. Furthermore, *peripheral* and *paranephritic lipomata* occur, sometimes primary with consecutive contraction of the kidney, and sometimes secondary with antecedent contraction of this organ. Finally, in rare instances, *lipoma of the pelvis of the kidney* is also observed. *Myxomata* have been met with as little knots, or under a larger form combined with sarcomata. A very interesting preparation of this kind, a "myxomatous sarcoma of the right kidney," of the size of a small apple, taken from a woman seventy years old, may be found in the collection of the Breslau Pathological Institute.[1] I will give a brief notice of an interesting case of *lipoma of the entire kidney* which I observed in All Saints' Hospital in Breslau.

---

[1] A. C. 1866. No. 32.

The subject was an old woman who died of marasmus. The urine showed nothing striking and was free from albumen. The right kidney was healthy, the left was enlarged to more than double its normal size. The large fatty capsule on the left side was in striking contrast to the absence of a capsule on the right. The fact most worthy of note was that the entire kidney consisted of fatty tissue without a trace of kidney tissue. The cortical portion, clearly defined, constituted a sort of cloak to the pyramids, which were sharply separated from each other. The capsule coadhered inseparably to the yellow mass of fat. The blood contained in the organ was quite scanty. The pelvis and ureter were healthy.

Quite an analogous observation is to be found in Rayer.

Sarcomata in the kidney are not at all uncommon, occurring as a secondary neoplasm. Both round-celled and spindle-celled sarcomata have been observed. They generally present themselves as not very extensive tumors. Furthermore, sarcoma may occur as a mixed tumor, simultaneously with carcinoma of the kidney, which will be spoken of again below under the head of Renal Cancer. So far as present observations go, *primary* sarcomata appear to occur as rarely in the kidneys as in other glands (with the exception, at the most, of the salivary glands). Hahn laid before the Obstetrical Society of Berlin[1] a sarcoma of the right kidney as large as a child's head. It was obtained from a child ten months old, and had grown to this size within four weeks. During this time the secretion of urine had not been diminished. Unfortunately, it is not stated whether this was a primary sarcoma of the kidney. In the case of a renal tumor which Eberth designated as Myxoma sarcomatodes renum,[2] and which was obtained from a girl seventeen months old, it remained undetermined whether it originated in the kidney or the supra-renal capsule. In cases of this kind, where the sarcoma attains such a size, it will constitute a tumor recognizable during life by palpation, and after the diagnosis of a neoplasm of the kidney has been settled it will especially be a question whether it is a sarcoma or a carcinoma. Aside from exploratory puncture of the tumor, which cannot always be carried out for purposes of diagnosis, and which is only unreliable because mixed forms, consisting of both species of tumors, exist, in which microscopic examination of the matters obtained by puncture would lead to perplexity, there are certain tests which might here be applicable for purposes of diagnosis. First of all, it is to be regarded as highly probable that a renal tumor which is susceptible of palpation is a sarcoma, if it is developed together with or following sarcomata which are accessible to diagnosis, such, for instance, as melano-sarcoma of the skin and of the eye-ball. Smaller melano-sarcomata of the kidney which escape palpation may be suspected when the urine in such cases assumes a dark color, because this color is due to the black elements of sarcoma which are washed out.[1]

---

[1] Berlin. klin. Wochenschrift, 1872. p. 269.

[2] Virchow's Archiv. LV. p. 518.

Gliomata are described by Virchow as soft, white, very tender, translucent, little knots, running up to the size of a cherry. They are developed in the cortical portion, are deficient in blood, and are of a distinctly medullary appearance. They are distinguished from cancer by the absence of epithelial elements.

Angiomata affect the blood-vessels in part, and in part the lymphatic vessels. Hæmatangioma cavernosum shows quite the same conditions of structure as teleangiectasia of the liver, with which it has also sometimes been observed to coexist. Virchow has found it most commonly at the surface, immediately beneath the capsule, occasionally in the uppermost portion of the medullary substance. It is generally found in the form of encapsulated lumps, varying in size from that of a cherry-stone to that of a walnut. *Lymphangioma* of the kidney has been described by Heschl in particular. Klebs considers these as cases of adenoma. They are of no practical interest. The formation of *granulation tissue* takes place in *syphilitic nephritis*. I have already described the most frequent form of syphilitic disease of the kidney under the head of Amyloid Degeneration (p. 621). True *gummy tumors* of the kidney, such as often occur in the liver in constitutional syphilis, have thus far been encountered but rarely, and in those few cases the nodules have almost always been quite small. Among the few instances in which large gummy tumors have been found in the kidney, I will first mention the observation of Cornil,[1] who claims once to have found a gummy tumor of the kidney, similar to those of the testicles and liver, in a case of amyloid degeneration of the kidneys dependent on syphilis. The largest tumor of this kind seems to be the one described by Moxon.[2] He found a gummy tumor the size of a small potato in the left kidney of a syphilitic woman. The growth had a regular surface, and consisted of a yellowish white substance, of quite uniform appearance, and of a firm, hard, and dry consistency. This was embedded in a large, white, lardaceous kidney.

---

[1] Compare *Eberth*, Virchow Archiv. LVIII. p. 58.

The observation made by Klebs also seems worthy of notice. He found in the left kidney pale, whitish, round collections, the centre of which presented a dead-white spot. On making a section through one of these, a tough, mucilaginous mass, two-fifths of an inch wide, was found to occupy the entire thickness of the cortical substance. The collections consisted of connective tissue, rich in cells, the cell elements being partly of the short, spindle-shaped variety, partly round, and at some points fatty. The urinary tubules had mainly disappeared; here and there they could still be demonstrated, though in an atrophied condition.

The conditions next to be considered are, at least in part, of greater practical significance than the neoplasms thus far described.

Renal Cysts and Cystic Degeneration of the Kidneys.

LITERATURE—*Bright*, Guy's Hosp. Rep. 1839.—*Frerichs*, Brightsche Krankheit, p. 28, and Colloidcysten der Nieren. Göttinger Studien. 1857. 1. Abthlg.—*Virchow*, Gesammelte Abhandlungen, p. 837, 864, also Geschwülste I. 270 und III. p. 94.—*Heusinger*, Angeborene Blasenniere. Marburg. 1862.—*Folwarzny*, Würzb. med. Zeitschrift. I. 1860, p. 151.—*Beckmann*, Virchow's Archiv.-IX.—*Erichsen*, ibidem, XXX.—*Hertz*, ibidem, XXXIII.—*Joh. Klein*, ibidem, XXXVII.—*Brueckner*, ibidem, XLVI.—*Koster*, Nederl. Ark. II. III. (Virchow-Hirsch

---

[1] Journal de l'anat. et phys. 1865. p. 97.

[2] Guy's Hosp. Reports. 1868.

Jahresber.) — *Ranvier*, Journal de l'anatomie et physiol. 1867, p. 445.—*Schlenzka*, Dissert. inaugural. Greifswald, 1867, and the literature given on page 543.

Pathology.

I propose to take up separately the questions of *cyst formation in extra-uterine life* and *congenital renal cysts.*

The development of cysts during extra-uterine life occurs, first, in apparently *healthy* kidneys. These cysts vary in size from that of a pin-head to that of a pea or even a walnut, projecting with a portion of their circumference above the surface of the kidney. They are either diffuse or arranged in groups, and are very thin-walled, so that a certain number of them burst on peeling off the capsule.

The contents of these cysts are generally clear, slightly yellowish, and contain albumen. They have been found to contain uric acid and carbonate of lime in small quantities, as well as colloid masses in the form of irregular lumps of various sizes, which give the reactions of albumen. These colloid masses are to be found even in the smallest cysts; the fluid, albuminous ingredients appear only in the larger ones, wherein cholesterine may also be discovered. Undoubtedly, in all cases, the urinary tubules take the most prominent part in the development of these cysts, inasmuch as it is altogether probable that the starting-point in their formation lies in the obstruction of the tubules with fibrinous masses. These cysts generally have no walls of their own, but are surrounded by unaltered interstitial tissue.

The *second* class of cysts developed in *extra-uterine life* are distinguished, to begin with, by their smaller size, rarely exceeding that of a pea, and accompanying interstitial proliferations of connective tissue, with or without enlargement of the kidney as a whole. They are most frequently found in the cortical portion. They are often developed out of the urinary tubules, have thick walls, and may then be recognized by their being strung together like beads on a string. The smaller ones are entirely filled with colloid masses. Others originate in the glomerules, and the remains of the vascular coil may be recognized within the dilated capsule, which is filled with colloid material and often with cell-masses and pigment-masses. The theory of the development of cysts out of the interstitial connective tissue, as was claimed by Erichsen and Hertz, has not thus far won any further recognition.

These cysts do not become the subjects of clinical observation unless, as is exceptionally the case, they attain to a very large size. In one instance, occurring in a woman sixty years old, the kidney was found to contain a cyst which was at least of the size of two fists, and which did not communicate with the calyces or pelvis of the kidney. Some doubt was entertained during life as to whether it might not be the distended gall-bladder.

In isolated cases *complete cystic degeneration of both kidneys* may be observed in adults. The kidneys are then more or less enlarged, and may attain such dimensions as to be felt during life. Both kidneys are found attacked, though perhaps not to an equal degree; the substance of the kidneys is transformed into a series of closed cysts which are imbedded in an abundance of connective tissue, and which vary in size, sometimes attaining to the dimensions of an orange. They contain tough, yellowish or reddish serum, or sometimes a gelatinous substance; their contents are always albuminous, but free from urinary substances, and are found to contain blood-corpuscles, pus-corpuscles, and cholesterine crystals. In very advanced cases every trace of kidney tissue is wanting; often, however, remnants of it remain in the midst of the connective tissue. The bladder, ureters and pelves of the kidneys are generally healthy. According to the reports now on hand these forms, too, appear to originate in dilated urinary tubules. The cysts are lined with simple epithelium, consisting of polygonal flat cells. It is not yet demonstrated whether the development of these cysts is connected with any congenital conditions.

The clinical history of this affection is as yet imperfectly studied. Most of the cases where such kidneys were met with were in persons between the ages of fifty and sixty, the youngest being thirty years old. Twice as many were men as women. The symptoms are not very characteristic. The course of the disease is chronic. No diminution in the urine appears, but during the advanced stages its specific gravity seems to be considerably lowered. Albuminuria and hæmaturia, recurring from time to time, are the most constant symptoms. As a general rule the course of the disease is latent and insidious, and if patients do succumb to the kidney affection, death generally follows suddenly with uræmic coma and convulsions. A kidney of this kind is to be found in the Institute of Pathological Anatomy in Breslau.[1]

It belonged to a woman, sixty-four years of age, who was made the subject of a judicial post-mortem, and who died of hemorrhage, the result of rupture of the posterior wall of the left auricle. The heart was considerably enlarged, the mitral and aortic valves were thickened, calcareous, and insufficient. Both kidneys were of the same size (15 × 9 × 4 cm. or 6 × 3.6 × 1.6 inches), and showed, over their entire surface, cysts from the size of a millet seed to nearly the size of a walnut, which were principally developed in the enormously enlarged cortical substance. Between the cysts there was still apparently much unaltered parenchyma, which, unfortunately, could not be more accurately examined on account of advanced putrefaction. The capsule was removed pretty easily and without injuring it. Some of these cysts contained in part a clear serous substance, not coagulable in alcohol, in part a brownish substance which coagulated to a firm mass in alcohol, while others of them contained almost purely bloody masses. In the liver there were extensive cicatricial lines and a series of cysts on its surface, from the size of a millet seed to that of a pea, one being as large as a hazelnut. The labia pudendi showed old cicatrices.

With reference, now, to *congenital cystic kidneys*, they are from three and one-half to six inches long, by from two to four inches wide and thick. In their size and their development they present great similarity to the cystic kidneys of adults. It does not seem probable that the latter are carried over from the fœtal period, because the individuals in question have generally died at a pretty advanced age, and experience teaches that only light grades of this disease are tolerated after birth. We are not as yet in possession of a sufficient number of observations to determine the question of whether these light grades are developed to higher ones after birth. As to the contents of the cysts, those of the smaller ones, at least, are found to be mingled with urinary matters. The formation of the cyst, therefore, depends on a retention of the secretion. The development of the same is participated in by the urinary tubules and the Malpighian capsules. With regard to the origin of this retention of secretion, it depends, according to Virchow, on an embryonic nephritis, which causes numerous narrowings of the urinary tubules, and leads to atrophy of the renal papillæ and obliteration of the pelvis of the kidney. Koster, on the other hand, finds the cause of congenital cystic kidneys in a primary lack of development of the urine-conducting apparatus. At the same time he takes no notice of the cases with open renal pelves and ureters, which cannot be explained on any other theory than that of Virchow, a series of valuable practical points concerning which are given in the statements of Kupffer with regard to the development of the urinary tubules. Inasmuch as a disconnected development of the urinary tubules and the pelvis of the kidney takes place in the fœtus, it is easy to understand how under some circumstances their connection remains imperfect.

---

[1] Acc. Catal. 1867. No. 62.

The development of this cystic kidney appears to be followed by grave consequences to the fœtus, too, as most of these children are born prematurely. If they are carried to full term, life can only be maintained, as was stated above, in case the development of these kidney changes is but slight. The higher grades of change, on the other hand, either constitute an absolute obstacle to birth and render embryotomy necessary, or, at least, cause so serious a distention of the abdomen, that the movements of the diaphragm are impeded, and breathing is rendered impossible.

It is a noticeable fact that these cystic kidneys are not infrequently combined at the same time with other congenital disturbances, constituting an argument in favor of the origin of the former in mechanical disturbances acting from without. The most interesting case of this kind was observed by Heusinger. It presented a cystic kidney on the right, together with absence of the right lower extremity and the right half of the female genitals, while nothing abnormal was found on the left side of the body. The occurrence of congenital cystic kidneys in children of the same mother also appears remarkable, though it is still more singular that children with cystic kidneys and healthy children should be borne alternately by perfectly normal and healthy mothers.

The latter circumstance, as well as combinations with other vices of primary formation, would lead us in such cases to regard the development of cystic kidneys as errors of development. The increased connective-tissue formation may just as well be a consequence as a cause of the cyst development, the purely mechanical conditions of closure of the urinary passages being sufficient to account for their genesis. At the same time Klebs has also found this increased development of connective tissue to be lacking.

Aside from congenital cystic degeneration of the kidneys, with enlargement of the kidneys, the same may also take place with shrinking of these organs. An interesting instance of this kind is to be found in the museum of pathological anatomy at Breslau.[1]

The left kidney is of the size of a bean, the right half as large. Neither of them shows any trace of kidney substance, but both are composed of a number of little cysts, in which, under the microscope, cells may be recognized which have undergone colloid degeneration. The muscular coat of the bladder is thickened. Cellular hypertrophy exists on the mucous membrane, especially of the fundus. The mouth of the right ureter is distinct, but the latter is closed above; the left ureter is free; both run a straight course. The bladder is the seat of an abscess. There is no urethra. The testicles are in the abdominal cavity. Talipes varus of the left foot.

The study of renal cysts may naturally be followed by that of *dilatation of the ureters and of the pelvis of the kidney, with consecutive disappearance of kidney tissue.* This is designated as

Hydronephrosis,

and is of great practical significance.

Literature and History.

The literature referred to on page 543, and furthermore:

Albers, Beobachtungen aus dem Gebiete der Pathologie. 1836. I. p. 40.—*Virchow*, Verhandlungen der Würzb. med. Ges. V.; also Geschwülste. 1863. I. p. 267; also Gesamm. Abhandlungen. 1856. p. 812.—*Todd*, Clinical Lectures, etc.

---

[1] 1869. Obd. Prot. 103.

London. 1857.—*Kussmaul*, Würzb. medic. Zeitschr. IV. 1863. p. 42.—*Saeringer*, Prager Vierteljahrschrift. 1867. I.—*W. Krause*, Langenbeck's Archiv. VII. p. 219.—*Spencer Wells*, Med. Times. 1868.—*Cooper Rose*, ibid.—*Ackermann*, Deutsch. Archiv. f. klin. Medic. I. 456.—*Heller*, ibid. V. p. 267. VI. p. 276.—*Hotz*, Berl. klin. Wochenschr. 1869. Nr. 23.—*Hildebrand*, Volkmann's Sammlung klinischer Vorträge. Nr. 5.—*Gusserow*, ibid. Nr. 18.—*E. Fraenkel*, Tageblatt der Breslauer Naturforscherversammlung. 1874.

Although the term "hydronephrosis" was first used by Rayer, the affection itself was very well known to the earlier observers. Thus, Friedrich August Walter, in the year 1800 (l. c.), not only gave a very striking description of the same, but also some historical notices thereof. He says that a number of writers before him allude to a similar disease of the kidneys. Prominent among them is Ruysch, who sometimes designates it as *expansio renum*, sometimes as *hernia renalis.* Walter himself called this condition *hydrops renalis*, while at the present day hydronephrosis is its customary designation. Indeed, it was formerly no uncommon thing for renal cysts to be confounded with this affection.

The clinical history of hydronephrosis belongs to the most recent periods, and, in fact, dates especially from the time when modern surgery embraced within its domain the operative removal of abdominal tumors. Since that time errors of diagnosis, especially the confounding of hydronephrosis with ovarian cysts, have become a matter of no little consequence. The question will hereafter be discussed of how far the diagnostic aids now at our command will enable us in future to avoid such errors.

Etiology.

Hydronephrosis—dilatation of the pelvis of the kidney and of the ureter, and subsequent more or less extensive disappearance of the renal parenchyma—is developed when obstacles are presented to the escape of the urine, and this fluid can only be evacuated incompletely or not at all. This obstacle to the evacuation of urine may lie in any division of the urinary passages, in the pelvis of the kidney, the ureter, the bladder, or the urethra. The obstacles may be either *congenital* or *acquired later in life*. The former, then, are caused by a vice of primary formation, and are often associated with other malformations, such as atresia ani, club-foot, harelip, etc.

The causes of *acquired* hydronephrosis are exceedingly multifarious, so that it would hardly be possible to arrive at a complete enumeration of them. The following are the most noteworthy: Concretions, which are wedged into the pelvis or the ureter, are tolerably frequent causes of hydronephrosis. The fact is not to be overlooked that sometimes, on making a post-mortem examination, the concretions which have caused the hydronephrosis are no longer to be found. They crumble in course of time, aided perhaps by the pressure of the column of fluid, and the fragments are discharged. The presence of small concretions gives us no right to conclude that calculi were really the inducing cause, as they may also be secondary formations. Nephrolithiasis is more frequently the cause of pyelonephritis than of hydronephrosis, though the two conditions are often combined. Among the rare causes of hydronephrosis may be mentioned anomalous or supernumerary renal arteries, which by reason of their peculiar course, twining around the ureters, compress them, and thus constitute a hinderance to the escape of urine.

In other cases narrowing or dilatation of the ureter may exist near its origin or termination, while everything points to some inflammatory or ulcerative process as the cause thereof; or bands resulting from parametritis may be present which bind the ureter to the margin of the pelvis. This fact naturally leads to the establishment of another fact, viz., that *in the female sex the ureters are liable to be narrowed in their calibre as the result of various pathological conditions of the genital apparatus.* Hence, hydronephrosis is more frequent in females than in males. Under this head may be classed compression of the ureters by *retroflexion of the pregnant as well as the non-pregnant uterus.*

The latter circumstance has of late been especially verified by Hildebrand, who has shown the mechanism by which in this way hydronephrosis of both sides may be brought about. The ureters, as we know, descend to the bladder on both sides alongside of the portio vaginalis of the uterus. If the middle portion of the uterus becomes bent at an angle, it may very easily happen that both ureters are similarly bent, and thus distorted downward and backward. Then the urine on its way to the bladder necessarily stagnates above the bend where it encounters an obstacle. It distends the ureter and the stagnation is continued upward as far as the pelvis of the kidney, and thus causes hydronephrosis. *Carcinoma of the uterus* is often associated with hydronephrosis. The narrowing or entire impermeability of the ureters in cancer of the uterus is very rarely due to a cancerous affection of these tubes themselves. It arises either through cancerous infiltration or cicatricial thickening of the pelvic cellular tissue—and this is the most frequent way—or through cancerous disease of the posterior wall of the bladder in the region of the trigonum vesicæ.

The following observation of Tuengel[1] may be briefly noticed as a curiosity in the pathogenesis of hydronephrosis: In a female body the kidney was found to be developed on one side only. This kidney had also been caused to dwindle by the pressure of its dilated pelvis and ureter. The cause of the hydronephrosis consisted in another and more rare error of development, viz., in the dilatation of one-half of a bifid uterus in which the menstrual fluid had accumulated by reason of atresia of the os uteri. Hydronephrosis may also occur as the result of *prolapsus uteri*. Virchow has shown that falling of the womb, even without descent of the fundus and due to enlargement of the cervix alone, may produce inversion of the vagina, which must result in displacement of the bladder. Inasmuch as the point at which the ureters enter the bladder is dragged down beneath the symphysis, where it is liable to be compressed and cause a damming up of the urine, a greater or less degree of hydronephrotic dilatation ensues according to the amount of obstruction to the escape of the fluid. *Ovarian and other tumors within the true pelvis* may also lead to narrowing of the ureters and subsequent hydronephrosis.

---

[1] Klinische Mittheilungen. 1860. p. 54.

This affection may also be induced by *abscess of the bladder*, which is liable to occur in either sex, whenever the abscess embraces in its domain the mouth of the ureter—a not infrequent occurrence. The condition here described is admirably illustrated by an interesting preparation in the Institute of Pathological Anatomy at Breslau.[1]

The case was that of a patient with stone in the bladder, for which a lateral lithotomy had been performed by Prof. Middeldorpff. At the fundus of the bladder, which showed a catarrhal affection of the mucous membrane and ulceration, there was an abscess of the size of a child's fist. The orifices of both ureters lay within the area of this abscess. The ureters were dilated to the size of a loop of small intestine, but not displaced. Their contents could still be emptied into the bladder by moderate pressure. Both kidneys were excessively enlarged, and appeared as irregular nodulated masses. The left kidney was completely transformed into a hydronephrotic sac, scarcely a trace of the renal parenchyma being present. The right kidney was less affected, though also greatly degenerated and hydronephrotic.

If *neoplasms of the bladder* occupy the orifices of the ureters, this may also lead to their dilatation and that of the renal pelvis. Such is particularly apt to be the case in carcinoma. *Strictures of the urethra*, as they are developed after gonorrhœa, rarely give rise to any considerable degree of hydronephrosis.

Aside from anomalies of the renal artery, as above referred to, and diverticula of the bladder, which are often congenital, the following *congenital causes of hydronephrosis* must yet be mentioned. First, certain anomalies of the ureter may exist at birth, preventing the escape of urine or rendering it difficult. Thus the ureter is sometimes found to consist of a solid cord ; at other times it enters the pelvis of the kidney at an acute angle, constituting a valve-like obstruction, which must necessarily be increased with the dilatation of the pelvis. A probe introduced from below readily enters the pelvis of the kidney, but the valve-like arrangement prevents the escape of urine. Sometimes stenosis of the lower portion of the ureter is encountered, producing the same effect. Steiner[2] reports a case of hydronephrosis in a child three and one-half years old, caused by a thick ring or welt encircling the vesical orifice of the left ureter. The latter was distended to the size of the small intestine. In this category may also be included cases of congenital abnormality of the urethra, such, particularly, as its complete or partial closure. Under such circumstances the bladder, ureters, and pelves of the kidneys are converted into extensive sacs.

---

[1] Obd. Protok. 1867. Nr. 66.

[2] Compend. der Kinderkrkht. 2d Edition. 1873. p. 320.

In the literature of this subject we meet with a series of cases where no etiological ground for the hydronephrosis could be demonstrated—that is, no hinderance to the flow of urine through the urinary passages. Nevertheless, it is evident that such a cause must have existed. The cause has either eventually disappeared, as, for instance, in case of concretions, as indicated above, or it has been overlooked in the autopsy, as may very easily happen in case of valvular arrangements or of obstruction due to cicatricial contractions about the uterus. These very readily escape observation as soon as the organs are once removed from their natural position and connections.

Pathology.

Pathological Anatomy.

The anatomical picture presented by hydronephrosis varies, first, according to whether the obstacle to the escape of urine is situated in the pelvis of the kidney or upper portion of the ureter, or in the lower portion of the same, or, finally, in some other part of the urinary passages. The lower down the obstruction is situated, the more extensive are the portions of the urinary tract embraced in the area of dilatation. In the slighter grades of hydronephrosis the pelvis of the kidney is only moderately dilated, the calyces participate in the dilatation, and the renal papillæ experience a corresponding flattening. In the higher grades, the distention of the kidney pelvis is more and more increased, while the parenchyma of the organ disappears in the same proportion. First the medullary substance atrophies; under the increased pressure of the confined fluids, the papillæ dwindle into scarcely recognizable prominences ; while the cortical portion as yet shows but slight changes. During the further progress of the affection this portion, too, atrophies, and is lost under the constant pressure to which it is exposed; and in the highest grades the kidney presents an enormous membranous sac, which, when filled with fluid, has a lobulated appearance. Such tumors may attain formidable proportions. They sometimes fill the entire half of the abdominal cavity, extending from the posterior portion of the diaphragm and the vertebral column to the upper outlet of the true pelvis, crowding away the liver, spleen and intestines, and forming numerous adhesions to the neighboring organs. Frequently, but by no means always, portions of intestine, especially the colon, lie on the hydronephrotic sac; in other cases the colon lies on the inner or outer surface of the same. The two are often found glued together, the tensely filled cyst compressing the corresponding portion of intestine. The wall of the sac, in hydronephrosis, consists of dense connective tissue with a smooth surface, the outer surface being made rough and uneven by a series of connective tissue adhesions, portions of tissue of a fibro-cartilaginous texture being sometimes found in this connective tissue. These tissues are rich in blood-vessels, taking their origin from the normal renal vessels. The external lobular appearance corresponds to a series of connective tissue septa in the interior of the sac, which themselves correspond to the greatly dilated calyces of the kidney, which open freely into the pelvis of the kidney and communicate with one another. Sometimes the dilatation only affects one or several of the calyces—then *partial* hydronephrosis exists, for instance, of one or two chambers. In the rare cases in which, there being two ureters, one of them is obstructed, the hydronephrotic sac also embraces only a portion of the kidney, which, however, does not prevent this partial hydronephrosis from attaining very grave dimensions. I have already stated that the cortical portion offers the longest resistance to atrophy resulting from pressure. Even in quite advanced cases we may often still find some islands of reddish tissue remaining in the membranous walls. Then the Malpighian bodies and the urinary tubules are found to be preserved in these remnants of tissue, the tubules containing fatty epithelium. Finally, however, these remnants of glandular tissue also disappear, and in the most extreme cases not a trace of it remains. If the obstacle is situated in the ureters, these are involved, to a correspondingly greater or less degree, in the process of dilatation. They become wider and longer. They are seen to attain the size of a man's thumb—this is very frequent—and even that of a small intestine, and by reason of their increased length they present a tortuous course. Their walls are sometimes thickened, particularly in those cases in which, before the occurrence of complete closure of the ureters, foreign bodies had passed through them, especially stones, and also in those in which the muscular structure of the ureters had been forced to overcome great obstacles.

The sacs of hydronephrosis contain fluids which, in general, represent a very watery, more or less modified urine. They contain urea, uric acid, the ordinary urates, and sometimes crystals of the oxalate of lime. Sometimes the fluid does not contain all the ingredients of normal urine; occasionally the uric acid is absent; urea is generally present, though often in but small quantities. Cooper Rose and Spencer Wells[1] have met with cases in which there was no urea. A part of the urinary constituents seems always to be reabsorbed. The reaction is often faintly alkaline, the specific gravity generally low, corresponding with the small amount of solid ingredients. The color of the fluid is not always the same; it may be a clear yellow, or turbid grayish yellow, or reddish to reddish brown, according as the urine is mingled with pus or blood. In the two latter instances the fluid would contain more abundant quantities of albumen, which is seldom entirely lacking. Epithelium is also now and then found in the fluid. In rare cases the contents of the sac consist of a thick, fatty fluid, or even of atheromatous or colloid masses. Hydronephrosis of the highest grade is generally unilateral, because the causes generally exist only on one side. If but one kidney is attacked, the other is generally found in a state of compensatory hypertrophy. If both kidneys are attacked, one is usually affected to a greater degree than the other. The causes of the hydronephrosis are determined by anatomical investigation (page 644 et seq.).

---

[1] Lancet, May 30, 1868.

Symptomatology.

The symptoms of hydronephrosis vary according to the grade of distention of the urinary passages, and according to whether one or both kidneys are involved. Slight degrees of hydronephrosis, whether unilateral or bilateral, which embarrass the flow of urine to but a limited extent, cause no symptoms during life. If the obstruction becomes more considerable, a double set of symptoms arises. On the one hand, those symptoms appear which are liable to accompany embarrassed, diminished, or arrested activity of the kidneys. This happens when both kidneys, or a single kidney (the other being absent), are the seat of advanced hydronephrotic degeneration. In his case of bilateral hydronephrosis, which occurred in a woman twenty-two years old, Fraenkel (l. c.) observed *transient but total anuria.* This occurred three times, lasting on one occasion for two days, on another for one day, and the last time for twelve hours. In each instance it followed one of the three performances of bilateral puncture undertaken by Fraenkel, while at other times the quantity of urine was normal. Fraenkel considers it probable that the urine was poured forth into the hydronephrotic sacs, emptied by the puncture, first filling and distending these, and not being evacuated externally until the weight of the accumulated urine was sufficient to overcome the obstruction to its escape. This explanation is undoubtedly correct. The other set of symptoms referred to above consists in the objective recognition of the hydronephrotic sac, when it has attained a sufficient size, as a palpable tumor. Sometimes both conditions are combined. I have already stated above that in cancer of the uterus both ureters are very often involved in the pathological process in such a way that they are compressed, and that subsequent hydronephrosis is developed. If but one ureter is impermeable, this is hardly noticed, the healthy kidney vicariously undertaking the function of the diseased one. But when both ureters are distended, and the hydronephrotic dilatation and disappearance of renal parenchyma extends to both organs, then the symptoms of acute or chronic uræmia are sure to arise in course of time. During the past ten years I have had occasion to observe the patients suffering from cancer of the womb, in the All Saints' Hospital at Breslau, and possess notes of forty-nine cases. Among these the signs of acute uræmia, severe convulsions, transient amaurosis, etc., occurred three times. Thirty women died with the symptoms of chronic uræmia; these patients lay for days, weeks, and even months in a comatose condition before death ensued. Some few observers have seen death follow, in such cases, with violent convulsions, during an acute uræmic attack. Sometimes, in very lean patients with relaxed abdominal walls, it is possible to feel the tumor caused by the hydronephrotic kidney. In very many cases of uterine cancer hydronephrosis accelerates the fatal termination, which is inevitable from the nature of the fundamental disease. In hydronephrosis—the result of retroflexion of the uterus—it is now and then possible, when they have reached a high grade of dilatation, to feel the enlarged ureters above Poupart's ligament. Under these circumstances the tumor may attain the size of a child's head. Hildebrand has particularly studied these very forms of the disease. After the uterus has been lifted up with the sound, and a catheter has been introduced into the bladder, it is possible, by the aid of external pressure on the abdominal walls, to evacuate the urine, and thus to effect a material reduction of the tumor. Even in comparatively slight distentions due to the same cause, careful objective examination may give positive evidence, as is taught by a case of Hildebrand's briefly subjoined by way of illustration. In this instance no tumor could be felt on the most careful examination, while the patient lay upon her back. On examining her in the standing posture, a tumor could be felt alongside of and somewhat above the uterus, on the left side, which, when pressed against the finger in the vagina by the hand on the abdomen, revealed an elongated form, and appeared somewhat painful. At different examinations, however, it changed in form, size, tenseness and softness of its walls. When the flexion of the uterus was radically improved, the tumor disappeared for good. In a case of procidentia uteri, with great elongation of the cervical portion of the uterus, which had existed for many years in a woman in the lowest walks of life, I saw death ensue under symptoms of chronic uræmia. Post-mortem examination showed great dilatation of both ureters and destruction of the renal parenchyma.

The tumor caused by hydronephrotic distention of the kidney often attains a very large size. It is generally unilateral, as has already been stated under the head of anatomical relations. Its position corresponds, in general, to that of renal tumors, as will be more particularly set forth hereafter, in connection with the description of cancer of the kidney. The tumor lies in the region of the loins, extends to the vertebral column, often upward into the hypochondrium, downward into the iliac region, and forward to the umbilicus. Within these limits the hydronephrotic tumor occupies the ground, to a greater or less extent, according to its volume. The circumscribed inflammations which generally cause numerous adhesions of such tumors frequently give rise to drawing or stabbing pains. Aside from this, large tumors of the kind cause numerous troubles through their constantly increasing size. They finally compress the thoracic organs, interfere with the action of the diaphragm, and cause a high degree of dyspnœa. Here, too, as in other renal tumors, the colon does not always lie upon the tumor. According to Spencer Wells, in renal tumors of the right side it lies to their inner side, in tumors on the left the same, or in front of the tumor, while the coils of small intestine are crowded to one side. These tumors feel soft, may be more or less distinctly defined by palpation, and give a dull percussion sound. They communicate a distinct sense of fluctuation, and often show a lobulated surface. Now and then the tumor diminishes in size with the escape of a large amount of urinous fluid through the bladder. The tumor is not movable, is generally painless, causes no feeling of inconvenience aside from that connected with its weight and a feeling of tension. It is often accompanied with obstinate constipation, especially if the colon is compressed by the tumor. If the disease is caused by nephrolithiasis, then well-marked attacks of renal colic are sometimes associated with it; and then, too, the urine may occasionally be bloody, whereas in hydronephrosis this fluid presents no pathognomonic signs, although it sometimes contains small quantities of pus.

Complications.

Hydronephrosis of one kidney is sometimes complicated with various diseases of the other kidney, which alone is responsible for the secretion of urine. It is very evident that such a complication involves the highest degree of danger.

Diagnosis.

Under some circumstances the diagnosis of hydronephrosis is easy, under others it is difficult or quite impossible. The lighter grades are not susceptible of diagnosis during life; in such cases the presence of hydronephrosis can only be conjectured with a certain degree of probability. If, for instance, uræmic symptoms arise during the course of uterine cancer, there is considerable justification for the diagnosis of this condition, based on the experience of analogous cases. This is still further confirmed if it is possible to feel the hydronephrotic sac. A diagnosis can likewise be made without much difficulty when it is possible, as in the cases of retroflexion of the womb reported by Hildebrand, to empty the tumor through the bladder, by pressure from without; or when a tumor in the abdomen disappears with the evacuation of a large quantity of urinous fluid. Frequently, however, insuperable obstacles are interposed to the making of a diagnosis. Hydronephrosis is most liable to be confounded with ovarian cysts, ascites, and hydatid cysts of the kidney. The point of greatest interest lies in distinguishing hydronephrosis from ovarian cysts, inasmuch as the question immediately arises with regard to the performance of ovariotomy. It is particularly easy to confound these two conditions, because both kinds of tumor give such distinct fluctuation. Here the question of the first development of the tumor is, naturally, of the greatest consequence, ovarian tumors always originating within the true pelvis. At the same time, every physician of experience knows how much or how little reliance is to be placed on the statements of the patient. In these cases the best service is rendered by the complete rectal examination, after Simon's method, for the purpose of proving whether the tumor can be isolated from, and be shown to be independent of, the uterus and ovaries. Based upon the authority of Spencer Wells, great weight has been assigned to the position of the intestine in renal cysts. Hotz even claimed that if the colon lay behind the tumor the condition might be supposed to be that of hydronephrosis in a horse-shoe kidney. But the position of the colon is by no means so constant, and it has repeatedly been found to lie to the posterior and external side of the tumor. On the other hand, cases of ovarian tumor have been known in which loops of intestine lay between the abdominal walls and the tumor. Far greater significance is to be attached to *exploratory puncture* and the examination of the fluid evacuated. If the cyst contains urinary constituents, this of course argues in favor of hydronephrosis. But cases exist, in the literature of the subject, in which no urinary constituents whatever could be found in the fluid of hydronephrosis, or where they existed in such small quantities that a very careful examination was necessary in order to detect them. For a time it was thought that the paralbumen discovered by Scherer was an integral and characteristic constituent of ovarian fluid. But it was afterwards not only found in peritoneal fluid, but Esmarch even discovered it in the fluids of hydronephrosis. It is evident from what has been said that the differential diagnosis between ovarian cysts and hydronephrosis may be involved in the greatest difficulties. Hydatid cysts of the kidney are only to be distinguished from hydronephrosis when the sac ruptures into the pelvis of the kidney—that is, when single cysts are passed with the urine. So far, however, as the differential diagnosis between different renal tumors is concerned, we must refer our readers to the sections devoted to this subject. Careful percussion generally guards us against confounding this condition with ascites. In unilateral hydronephrosis the matter is tolerably simple. Dullness is then found only on one side of the abdomen, while in ascites dullness is demonstrable in both lateral portions of the cavity. Bilateral hydronephrosis is distinguished from ascites by the fact that in the former the dullness persists when the patient is laid on his side, whereas in ascites the dullness disappears from the highest point.

Duration, Termination, and Prognosis.

In congenital hydronephrosis, life is seldom long maintained. The fœtus is either born dead or dies soon after birth, as the result of the crowding of the thoracic organs by the enormously distended abdomen.

The duration of acquired cases is very variable, and in most cases can hardly be measured, even approximately, on account of the impossibility of fixing the beginning of the malady. It depends, furthermore, upon the nature of the fundamental disease and the grade to which the affection has attained. If the obstruction to the escape of urine is slight, if the hydronephrosis is unilateral and the other kidney is healthy, the duration of the affection may be quite long. The terminations of hydronephrosis are seldom favorable; in the best cases a cure is accompanied with defects. The most frequent cures are observed in cases arising from the presence of calculi. If these are discharged, the contents of the hydronephrotic sac can escape, the passage being now open; and if no new accumulations take place from any cause, the sac may waste away. Furthermore, those cases of hydronephrosis caused by retroflexion of the uterus may also terminate in recovery after removal of the fundamental disease. In general, however, the prognosis is unfavorable, because the causes of hydronephrosis usually consist of irreparable injuries, which induce a fatal termination, partly on their own account and partly through the ever advancing destruction of the renal parenchyma.

Therapeutics.

We have seen that in some cases of hydronephrosis, which are developed as the result of nephrolithiasis, a spontaneous cure takes place. In the same way the cure of retroversion of the uterus, which has caused hydronephrosis, may be followed by the cure of the latter. On the whole, however, the science of therapeutics is helpless as over against the causes of hydronephrosis. Operative interference in this disease has been tried in various ways. The results thus far are not exactly encouraging, and, as a general rule, we shall be justified in having recourse to such means only when life is threatened, as, for instance, through mechanical compression of the thoracic organs. Roberts succeeded in one case, which occurred in a girl eight years old, by means of repeated kneading of a soft, fluctuating tumor in the abdomen, in effecting the evacuation of a large amount of urine, whereupon the tumor disappeared and did not show itself again while the little patient was under observation. It is evident that such manipulations may only be practised in the mildest manner, and that a favorable result would be the rare exception. The puncture of hydronephrotic sacs is dangerous, because an outbreak of general peritonitis is always to be feared. It is generally of little value because the sac is likely to refill in a short time. Hillier attained a temporary result in a case of congenital hydronephrosis which he had repeatedly punctured. Fraenkel's patient (compare p. 651, and according to a private communication) had already been punctured seven times by other physicians when she fell into his hands. He himself repeated the operation three times. After that the patient would not submit to the operation any more on account of its apparent inutility, the more rapid refilling of the sacs, the invariably high fever it induced, and the pain it involved. Threatened œdema of the lungs calls for the evacuation of the tumor. Dr. Maas proposed first to unload the right tumor by double puncture and subsequent incision, according to Simon's method, and, if this were well borne, about a week later to operate on the other. Maas thrust two trocars into the right tumor three centimetres apart, and *immediately* closed the stopcocks. The canulas remained in position four days without any special reaction. On the third day there was a flow of urine alongside of the canulas. On the fourth day there was a heavy chill. An incision was immediately made through the walls of the abdomen (layer by layer) and those of the tumor between the two trocar canulas, which were now removed. Drainage was employed, with carbolic acid dressings. The secretion of urine was maintained unhindered. Reaction was very slight. Death ensued on the sixth day after the operation, with evidences of increasing marasmus.

Such measures may only be ventured upon when the emptying of the hydronephrotic sacs is peremptorily demanded as a means of saving life.

Cancer of the Kidney.

Literature and History.

Aside from the general literature referred to on page 543: *Bright*, Guy's Hosp. Rep. London. 1839.—*Walshe*, The nature and treatment of cancer. London. 1846. —*Koehler*, Krebs und Scheinkrebskrankheiten. Stuttgardt. 1853. p. 415.—*Todd*, Clinical lectures on certain diseases of the urinary organs, etc. London. 1857. p. 42.—*Doederlein*, Inauguraldissertation. Erlangen. 1860.—*Wagner*, Archiv f. phys. Heilk. 1859.—*The same*, Arch. d. Heilkunde. 1860.—*West*, Lectures on the diseases of childhood and infancy. London. 1848.—*Kussmaul*, Würzburger medic. Zeitschrift. 1863. p. 38.—*Waldeyer*, Virchow's Archiv. Bd. XLI. and LV.—*Pereverseff*, ibid. Bd. LIX.—*Emile Neumann*, Essai sur le cancer du rein. Paris. 1873 (contains a tolerably full index to the literature of the subject).—Dissertations of *Doederlein* (Erlangen, 1860), *Eberhard* (Tübingen, 1869), *Jerzykowsky* (Breslau, 1871), *Michels* (Berlin, 1872), and numerous reports of cases.—*K. Schroeder*, Ein Fall von paranephritischem Carcinom. Kiel. 1874.

Cancer of the kidney, on account of its rarity, was comparatively late in attracting the attention of physicians. Whereas Baillie, in his celebrated series of engravings on copper, issued at the beginning of this century, gave a correct picture of phthisis renalis, nothing was said about cancer of the kidney. With the exception of a few observations that can be utilized, for instance, those in Wilson's Diseases of the Kidney, London, 1817, and G. Koenig's Abhandlung über Nierenkrankheiten, Leipzig, 1826, nothing of any value is to be found in literature until after the year 1830. It is true that at the present day we shall be able to make no use of Koenig's refinements of diagnosis, whereby he undertook to distinguish, during life, between scirrhus, steatoma, fungus, and medullary sarcoma of the kidney. This form of disease was placed upon a more substantial basis by Cruveilhier, in his Pathological Anatomy, in 1829, which was succeeded by the classical work of Rayer. Then followed the well-known treatises of Walshe, who gives a good synopsis of what had been accomplished up to that time, and of Lebert, who distinguished, better than his predecessors, between the primary and the consecutive or secondary forms of cancerous disease of the kidney. More recent literature has furnished us with a tolerably rich history of cases which renders it very practicable to give a clinical portrait of primary cancer of the kidney. Secondary cancer of the kidney is scarcely ever the subject of clinical observation, and will here only be briefly touched upon in the description of the anatomical relations of the disease.

Etiology.

We have as little reliable information with regard to the true cause of renal cancer as we have concerning the pathogenesis of cancer in other organs. No hereditary predisposition to renal cancer can be demonstrated from the material in our possession. The disease is not congenital, nor are there certain generations in which cancer of the kidney has frequently occurred. On the other hand, one point is worthy of consideration, in connection with cancer of the kidney, to which Virchow, in particular, has called attention as bearing on the etiology of malignant tumors in general, viz., the matter of irritants of various kinds, mechanical, chemical, etc. The sheltered position of the kidney certainly preserves it far more than most organs from mechanical injuries, and we are not, at present, able to adduce strict proof that such injuries cause cancer; for it is always possible that certain manifestations—such, for instance, as hæmaturia from traumatic injury—may only have been the means of bringing into notice a previously existing carcinoma of the kidney. At the same time, the probabilities in favor of such a causal relation grow with the number of instances adduced in proof, and a few of these cases will therefore be given in brief.

Even Chomel, in 1829, refers to a cancer of the kidney, which was supposed to have originated from a blow. It grew so large as to destroy a part of the anterior wall of the abdomen. Bright gives the history of a young woman who, five months before, had given birth to a healthy child, and who died some months afterward of cancer of the right kidney. Three months previously she fell down stairs, and she dated her suffering from that time. Manzolini's[1] case was that of a boy, seven years old, who six months previously had received a kick in his left side—afterward having fever and hæmaturia. These symptoms subsided at the end of two weeks. The boy died of medullary cancer of the left kidney. W. Brinton[2] reports the history of a man forty years of age, a cook by occupation, who found blood in his urine after having received a blow, and two years later noticed a tumor in his abdomen, which afterward proved to be carcinoma of the kidney. The most interesting case of the kind was published by Jerzykowsky. A lady in the upper walks of life had fallen down several steps seventeen years before he saw her. The fall caused a contusion of the right side of the abdomen, as the result of which she for a long time experienced pain in the right hip, lumbar, and abdominal region. Immediately following the accident she suffered from slight hæmaturia, with intermissions of some weeks in length. Six months after the fall she noticed a slight swelling under the margin of the right ribs. One year before her death the tumor had attained immense proportions. She died eighteen years after the fall, of carcinoma of the kidney.

Still, even if in these cases the traumatic injury caused the development of carcinoma of the kidney, the question still arises why in one case nephritis is developed, in another perinephritis, and in a third carcinoma. These are riddles which we cannot at present solve, and which force us to adopt the doctrine of a so-called individual predisposition.

Primary cancer of the kidney is a rare affection, though not so rare by far as Tanchou claims. This writer found only three cases of primary cancer of the kidney among 8,300 cases of carcinoma which he obtained from the mortality lists of the Department of the Seine during the years 1830 to '40. The results given in the justly celebrated work of Marc d'Espine on the mortality statistics of the Canton of Geneva during thirteen years[3] are far nearer the truth than the not very credible statements of Tanchou. He found two fatal cases of cancer of the kidney among 889 deaths from cancer, that is, 0.3 per cent. Next to his figures come the statements of Virchow.[4] His data, resting mainly on the anatomical conditions found present, embrace the cases of mortality that occurred within four years in Würzburg from carcinoma, cancroid, and sarcoma. Half of one per cent. (0.5) of all the fatal cases of malignant neoplasms fell to the share of the kidneys. Willigk has collected the results of the post-mortems in the Institute of Pathological Anatomy of Prague.[1] He found 4.6 per cent. of the carcinomata met with to be renal carcinomata, secondary cancers being of course included in this count. The rarity of cancer of the kidney is further shown by the fact that Steiner[2] found only four cases thereof among 100,000 children in the Children's Hospital at Prague, and these were in children from three to five years of age, and yet there is no organ of the thoracic or abdominal cavity so often attacked during childhood as the kidney. In Frerich's clinique in the Charité Hospital at Berlin three cases of cancer of the kidney were registered in ten years.[3]

---

[1] Schmidt's Jahrbb. 94. p. 74.

[2] Schmidt's Jahrbb. 97. p. 150.

[3] Essai analytique et critique de statistique mortuaire comparée. 1858. p. 369.

[4] Beiträge zur Statistik der Stadt Würzburg. 1859, reprint, pp. 18 and 19.

The earlier authors believed cancer of the kidney, during the age of childhood, to be a curiosity, and that renal cancer, like that of other organs, was quite distinctively a disease of advanced life. More recent works, based upon a larger experience, teach that cases of cancer of the kidney are distributed to two periods of life, early childhood up to the age of about five years, and old age, while the periods of youth and vigorous manhood are far less frequently attacked. Cancer of the kidney is the most frequent cancer of childhood. Hirschsprung[4] found the kidney attacked fifteen times among twenty-nine cases of cancer in children. I have collected sixty-one cases of primary cancer of the kidney from literature. In nine of these the age is not given, the remaining fifty-two are distributed as follows. First of all, we notice among them twenty children under the age of ten, the remaining thirty-two being more or less grown up. The details appear in the following table:

Under 1 year.	1 to 5	5–8	16	21–30	31–40	41–50	51–60	61–70
5	11	4	1	3	6	6	10	16 cases.

---

[1] Schmidt's Jahrbb. 1856. Bd. XCII. p. 285.

[2] Compend. der Kinderkrankheiten. 2 Aufl. 1873. p. 318.

[3] *Michel's* Inaug. Dissert.

[4] Virchow-Hirsch Jahresber. 1868.

While, as a general rule, cancer is more frequent among women than men, which is to a great degree owing to the frequency with which the female genital organs are attacked, cancer of the kidney, like that of the œsophagus and bulb of the eye, is most prevalent among men, a fact which was noticed by Marc d'Espine and Lebert. All later authors agree in this. In fifty-six cases in which the sex was given I found thirty-eight men and eighteen women. Still, this predisposition on the part of the male sex seems to be confined almost exclusively to the more advanced years. For amongst fifteen children there were eight boys and seven girls, and among thirty-two adults, twenty-three men and nine women.

Pathology.

Pathological Anatomy.

Carcinoma in the kidney is observed partly as a primary and partly as a secondary neoplasm. The latter occurs not infrequently in the kidneys, sometimes as one of the manifestations of general carcinomatous disease, at other times as the result of the direct spread of cancer from some neighboring organ to the kidney. In the former case both kidneys are generally involved, in the latter but one. Secondary cancerous growths in the kidney seldom attain any great dimensions ; the size of a walnut is pretty considerable. On the other hand, the very small, miliary cancerous nodules, such as frequently occur in the liver, are very rare here ; the smallest that I have observed were from the size of a pin-head to that of a pea.

Primary cancer of the kidney is usually confined to one kidney, the right one being most frequently attacked, rarely both. Among 59 cases of cancer of the kidney which I have collected in literature, 31 involved the right, 23 the left, and 5 both kidneys. Klebs makes the statement—at variance with other observers—that the left kidney seems to be most frequently attacked. Cancerous kidneys generally attain twice or three times the size of normal ones. It is only exceptionally that kidneys with primary cancer present the volume of the normal organ. Sometimes cancerous kidneys attain an excessive size. Spencer Wells saw one in a child four years old that weighed between sixteen and seventeen pounds. The size of cancerous kidneys does not increase in direct proportion to the age of the individual attacked, but quite to the contrary. It is during the age of childhood, in particular, that we find not only comparatively, but almost absolutely, the largest cancers of the kidney. Their growth is sometimes very rapid. This extraordinarily rapid growth, while it is not exactly exclusively characteristic of renal cancer, is more frequent here than in the cancer of other internal organs. If one kidney remains free of cancer it generally becomes hypertrophied. In very rare cases it is attacked with amyloid degeneration. Even in cancerous kidneys themselves, under these circumstances, amyloid degeneration of the Malpighian bodies has been observed in portions that were free from cancer.

In a certain series of cases of renal cancer the neoplasm is distributed as a uniform diffuse deposit over the entire organ. The form of the kidney is then usually preserved, only the organ is somewhat more round and plump. On making a section thereof it is often still possible to recognize the boundary between the medullary and the cortical portions.

In other cases the cancerous kidney presents itself as a nodulated tumor covered with many larger and smaller protuberances. After the entire kidney, or the greater part of it, is involved in the neoplasm, the cancer pushes its proliferations into the pelvis of the kidney, and sometimes even into the ureters. While those renal cancers, which are characterized by a uniform infiltration of their entire tissue, present a homogeneous whitish or yellow surface on section, the form which is marked by a nodulated surface on section also shows certain nodules, which are more or less sharply separated from the surrounding tissue, and are sometimes distinctly encapsulated. The renal tissue lying between the nodules is sometimes healthy, often hyperæmic and loose, the interstitial tissue being often greatly increased. In the larger nodules softening very often occurs at certain points; furthermore, extravasations of blood are very likely to take place in renal cancers, as the result of the rupture of the numerous very thin-walled vessels. In some isolated instances cavities are encountered, up to the size of a man's fist, filled with pulpy, sometimes fœtid contents—detritus and shreddy masses—bearing the semblance of an abscess. On more accurate examination the detritus is found to consist of cells that have undergone granular and fatty degeneration, with here and there some fat-crystals. Until the most recent period the development and the origin of carcinoma in the kidney, too, were looked for in the connective tissue of the organ. Waldeyer urged against this theory that the cancer-cells, and the cancer-bodies of renal carcinoma developed from them, merely represent derivatives, non-typical proliferations of the epithelium of the urinary tubules. An objection raised to Waldeyer's view was that the endothelium of the blood-vessels and lymphatic vessels constituted a part of the proliferated epithelium. This objection has been quite recently overthrown by Pereverseff, who demonstrated that the cancerous epithelial cells were still enclosed in the tunica propria of the urinary tubules, and that within the same urinary tubules there were in one part normal and in another part proliferated epithelial cells, so that one could follow the transition from one to the other. The cancer stroma, he says, in the earlier stages is formed only of the tunicæ propriæ and the small amount of connective tissue uniting them. It was only in the larger nodules, where there were no longer any normal urinary tubules, according to his observation, that there was also a proliferation of interstitial connective tissue.

Carcinoma of the kidney appears either as a scirrhus or a medullary carcinoma, or as a simple carcinoma, which Waldeyer ranks between the two. These differences in external characteristics are dependent upon a greater or less wealth of blood-vessels and cells. For a rich development of blood-vessels always causes a powerful development of cancer-bodies. In soft, medullary cancer of the kidney it may happen that the framework consists entirely of thin-walled vessels, in some places without any adventitious tissue; while in simple carcinoma, and especially in scirrhus, the connective tissue framework is not only more abundantly developed, but may even be in the preponderance. At the boundary between the remnants of kidney substance and the neoplasm a layer of connective tissue is interposed—as is often the case, for instance, in the liver. If one examines a simple carcinoma of the kidney more carefully, he will see, along the line of division between healthy and diseased tissue, in addition to an increase of interstitial tissue, certain enlarged urinary tubules crowded full of large, darkly granular cells. We may not infrequently see groups of such altered tubules, with proliferating epithelium, lying alongside of one another in knots. Aside from these regularly arranged portions, we may elsewhere see roundish formations, with many side-sprouts growing in every direction and terminating in blind sacs. They are composed of cells which are entirely identical with those of the ordinary tubules. Robin's description of an epithelioma of the kidney (1855) shows great analogy with that here given, and Klebs has described transitions from simple adenoma to carcinoma, which he designates as adeno-carcinoma. The only criterion whereby we can distinguish between the two, histologically, is the non-typical proliferation of epithelial cells. By this means the so-called cancer-bodies of Waldeyer are formed. In the medullary form the abundant cells often cover the cancer framework (which always stands out more prominently in simple carcinoma and scirrhus), so that it is often necessary to brush them off before the alveolar structure can be seen. Sometimes, in one and the same cancer, soft and harder, and even scirrhous portions, alternate with each other. As a general rule, however, it may be stated that the majority of cases of renal cancer belong to the scirrhous variety. In the scirrhous forms certain spots will be found where a devastation of the renal parenchyma has taken place as the result of indurative proliferation of connective tissue. The connective tissue here is sometimes very poor in cells, and then contains many urinary tubules, obliterated through contraction or fatty degeneration; at other spots there is an abundant proliferation of small cells. Cases of scirrhous degeneration involving the entire kidney have been described by Cruveilhier, Walshe, and Lebert. In some renal cancers the Malpighian capsules are dilated and undergo cystic degeneration. Braidwood is the only writer who speaks of the cancerous degeneration of these bodies. He regards the hæmaturia, which is so frequent in renal cancer, as a result of the cancerous degeneration of the glomerules. Soft, medullary cancers of the kidney are often designated as "fungus hæmatodes." This is to be ascribed to the circumstance that cancers of the kidney, as well as of the liver and testicle, are so often rich in wide, thin-walled vessels. These sometimes show partial aneurismal dilatations (Cornil).

In some few cases primary cylinder-cell cancer is developed in the kidney. E. Wagner describes an instance of this in which secondary nodules, with the same arrangement of epithelial elements, were found in the liver. An interesting feature in this renal cancer consisted of a portion of the neoplasm, which was as large as an egg and as hard as a rock, and consisted of connective tissue that had undergone calcareous degeneration. Mixed tumors are also met with. Under this head may be classed those cases of renal tumor, two of which are described by E. Wagner, which, like the so-called syphonoma, cylindroma, etc., present a combination of cancer, sarcoma, and glandular tumor. In a primary renal cancer I lately found the framework to consist also, in part, of spindle-shaped cells that could be isolated, while in other portions of the same tumor the framework differed in no respect from that usual in cancer. In some, though it appears very rare, instances, colloid carcinomata have also been observed in the kidneys. Such cases are found in Gluge and Rokitansky. The latter observed this twice. Quite recently Schueppel[1] saw a cancer of the right kidney weighing ten kilogrammes (26 lbs. 8 oz.), which showed in part the characteristics of an encephaloid in a state of fatty degeneration, and in part those of an alveolar colloid carcinoma. It is difficult to determine whether the cases of cancer melanodes reported by Bright, Rokitansky, and Lebert were of a sarcomatous or carcinomatous nature. I am not aware of any more recent observations of melanotic cancer of the kidney.

Cancerous kidneys usually soon form adhesions to neighboring organs, which, as a rule, prevent their dislocation. In some instances this takes place, nevertheless, as was first observed by Troja. A scirrhous tumor of the kidney may sometimes leave its natural position, by reason of its weight, and may be felt, as a tumor, beneath the floating ribs. Some other instances, from more recent literature, are given in the section on movable kidney. In Robin's case of epithelioma of the kidney, the diseased organ lay upon the vertebral column, as if riding upon it. The adhesions referred to are developed by means of the neoplasm attacking the capsule of the kidney and the surrounding connective tissue, and spreading to the supra-renal capsule; but this is by no means always the case. At other times the fibrous capsule is only found to be much thickened, traversed by highly injected vessels. The cancer may also spread by continuity to the retro-peritoneal glands, more rarely to the liver, after adhesion of a cancerous kidney on the right side with the lower surface of this organ. Furthermore, adhesions sometimes take place to the intestines—for instance, with the colon passing over a carcinoma, or with loops of small intestine lying in the deeper portion of the abdominal cavity (Faludi). In some rare instances, in carcinoma of the right kidney, its attachment to a narrowed duodenum has been reported. Then compression of the latter may lead to dilatation of the stomach, as in a case of F. von Niemeyer's.[1] Rayer even reports a case in which the cancer perforated into the duodenum. Furthermore, perforation of the abdominal wall, in the case of a cancerous kidney attached thereto, has been reported, though, it would appear, only once (Abele).

---

[1] *Eberhard's* Dissertation.

This was in a girl three years old. On a projecting portion of the skin, corresponding to the renal neoplasm, a roseate inflammation of the skin was developed. It resulted in an ulcer, through which a growth sprouted forth during the night, bearing the characteristic marks of a medullary cancer. A piece of intestine protruded alongside of this, which became gangrenous, so that, for five days before the fatal termination, thin, brownish, fæcal matter poured from the opening.

Reference has already been made to the fact that renal cancer very often spreads to the pelvis of the kidney and the ureter. Aside from cancerous affections of the pelvis of the kidney, other anomalies of the same may take place in renal cancer. In Jerzykowsky's case a peculiar distortion of the pelvis of the kidney is described, from which ten to twenty prolongations, lined with mucous membrane, radiated in every direction, the calyces of the kidney not only being immensely distended, but very much increased in length. Now and then blood-clots are found in the pelvis of the kidney, sometimes deposited in layers like those of aneurisms. The cancer, which occasionally extends into the ureter, may entirely fill this up with cancerous masses. Sometimes also this canal is occluded with blood-clots, and in other cases it is compressed by cancerous masses from without.

---

[1] *Eberhard's* Dissertation.

The renal vein is as often altered, in cancer of the kidney, as the renal pelvis. The vein being surrounded on all sides by carcinomatous masses, is generally itself involved, rarely remaining free. The walls of the vessel are at first compressed, then gradually destroyed, and the calibre of the vein, little by little, is filled with cancerous masses. In this way the growth spreads farther, and sometimes reaches the inferior vena cava. This gives occasion for secondary cancerous infection of the lung through embolism. In one case Gintrac saw the vena azygos also filled with cancerous masses. As the result of renal cancer, therefore, secondary deposits in other organs take place, partly by extension of the neoplasm (in its continuity), partly as the result of general carcinomatous infection. Secondary carcinomata are developed in more than half the cases of renal cancer; in the smaller half the disease is confined to the kidney. The most frequent seat of secondary deposits is in the lymphatic glands of the hilus of the kidney, the retroperitoneal and the mesenteric glands. Sometimes, as the result of carcinomatous disease of the kidney and of the lymphatic glands lying in its neighborhood, very large tumors are developed, in which it is impossible to decide where the neoplasm originated (as in Doederlein's case). According to Waldeyer's doctrine, which claims an epithelial origin for all carcinomata, we should have to refer, for the primary starting-point of the cancer in all such cases, to the organs containing epithelium—the kidneys. On the other hand, Zenker, and quite recently Karl Schroeder, have called attention to the existence of a peculiar variety of carcinomata of the kidney, which are designated as paranephritic, and which are characterized by the fact that, according to their external location, they appear as renal carcinomata, but that they do not arise from the kidney itself, but in its immediate vicinity—at the hilus, indeed; that they immediately penetrate into the capsule of the kidney, and, extending their growth inside of the same, secondarily destroy the kidney. Schroeder is inclined to the opinion that such growths originate in the endothelium of the blood-vessels, and that, accordingly, they should be distinguished from true epithelial carcinomata.

The lungs are almost as often the seat of secondary cancerous disease as the lymphatic glands above alluded to. This appears to be occasioned by the transportation of cancer elements to the lungs through the current of venous blood—a circumstance to which Budd called attention.[1] The liver is also often secondarily diseased. The supra-renal capsules and the heart are comparatively rarely attacked, still more rarely the vertebral column and the ribs, and most rarely of all the pleura and mediastinum. The latter took place in a case of Todd's, where life was destroyed by a hemorrhagic pleuritic exudation, the result of secondary cancer of the pleura and mediastinum following on cancer of the kidney.

It is very remarkable that, in spite of their anatomical and functional relations, renal cancer is very seldom accompanied by cancer of the lower urinary passages. On the other hand, the frequency with which cancer of the testicle is followed by renal cancer is also striking. One of F. von Niemeyer's patients had carcinoma of the testicle, which he thought originated from the blow of a whip, while he attributed a simultaneous carcinoma of the kidney to the pressure of a heavy purse.[2] Fleming[3] observed a case of cancer of the prostate in a man sixty years old; the bladder was healthy, but there was cancer of both kidneys.

So far as other anomalies are concerned that occur as the result of renal cancer, renal calculi have been observed in a certain series of cases, occurring sometimes in the healthy and sometimes in the cancerous kidney.

The other abdominal organs generally undergo changes of position. The colon is commonly found slack, empty, and collapsed in front of the tumor; the small intestines are crowded over to the opposite side. If the right kidney is diseased, the liver is pushed over to the left, often turned on its transverse axis, so that the upper surface assumes a vertical position and applies itself to the arch of the ribs and the abdominal wall. This is especially true when, as in Doederlein's case, the tumor proceeds from the upper end of the kidney and grows in the direction of the right hypochondrium. If the tumor originates in the left kidney, the stomach is pushed to the right, and the spleen moves high up into the arch of the diaphragm. More rarely a movable spleen has been observed in connection with renal cancer. In one case of Roberts', the spleen could be felt in the iliac fossa. If the renal tumor attains very large proportions, the thoracic viscera may also be compressed.

---

[1] On Diseases of the Liver. London. 1845.

[2] *Eberhard's* Dissertation.

[3] Dublin Journal. Aug. 1867. p. 235.

Symptomatology.

The symptoms of renal cancer, like those of cancerous degeneration of a number of other internal organs, are at first very obscure. Sometimes the first symptom is *pain* in the region of the loins. This is generally at first very insignificant, is often little regarded by the patient, and the physician has not the necessary data for determining its significance. The most important symptoms to be considered are the *renal tumor* and the *hæmaturia.* The latter may be observed at an early stage of the disease. But we must premise the statement that secondary cancer of the kidney is hardly ever the object of clinical observation. Still, even primary cancers sometimes run their course without giving any symptoms. I observed such a case in Breslau. It occurred in a woman of advanced years, who died of mitral insufficiency. At the autopsy both kidneys, but especially the left, were found to contain a moderate number of hard, white nodules, the largest of which were of the size of a chestnut. The organ was scarcely enlarged. The urine had never been altered. In the liver there were some nodules which varied from the size of a pea to that of a cherry. At both points, the liver and kidney, the disease was simple carcinoma.

Hæmaturia is sometimes the first symptom of renal cancer. It is often developed without any preceding pain. It is often impossible to make much out of this symptom if it is quite isolated, though a renal hemorrhage, arising without any external cause and running its course without pain, is always to be looked upon with suspicion. The matter assumes a more definite shape if another very common sign presents itself, viz., *a tumor of the kidney.* Roberts goes so far as to assert that in every case of renal cancer that terminates fatally, one or both of these symptoms—hæmaturia and a tumor—must be present. This is certainly claiming too much, although exceptions to the rule are rare. Among the exceptions are my own case just narrated, Flemming's observation, and one by Hirtz. In the latter case the only disturbances were uncontrollable diarrhœa, increasing marasmus, and œdema of the legs. The tumor, in particular, is one of the most constant signs of renal cancer, and, at least in the later stages, it usually attains such a size that it could not escape the notice of a careful observer. It is especially worthy of mention that in children cancerous tumors of the kidney may attain positively enormous dimensions. They represent, as a rule, the largest tumors that occur in children, especially among abdominal tumors. They generally begin in the lateral region of the abdomen, the loins, between the lower ribs and the crest of the ilium, and then grow upward, but more particularly downward and forward toward the pubes and the navel. It would be a great mistake *always* to expect to find increased dullness in the region of the loins in case of a renal tumor. On the contrary, cancerous neoplasms of the kidney seem more prone to involve the anterior portion of the organ, where the soft, yielding intestines offer far less resistance than the muscles of the loins, but at the same time prevent the occurrence of dullness. The position of the tumor and the signs of displacement of neighboring organs will vary according as the cancer involves the entire kidney, or its upper or lower portion. The tumor is first observed between the lower ribs and the crest of the ilium; in course of time, however, it extends forward to the navel, upward into the left hypochondrium, downward into the iliac fossa. In rare instances the tumor fills the entire abdomen. Percussion elicits a dull tone only if the tumor lies against the wall of the abdomen. But if coils of intestine lie between them, the tone is more or less of a dull tympanitic character. The position of the intestines in their relation to the tumor demands special attention. In cancer of the right kidney the small intestine is crowded to the left, the cæcum and the lower part of the ascending colon are generally to be found at the outer side of the tumor, while the upper part of the ascending colon is raised up and runs obliquely from right to left in front of the diseased organ. In cancer of the left kidney the descending colon, and sometimes a part of the small intestine, almost always lies in front of the tumor, separating it from the abdominal wall. Much importance has rightly been attached to the position of the intestine in front of the tumor, and it should at once be carefully borne in mind in making an examination. If the organs are in their natural position this usually enables us to distinguish a tumor of the kidney from that of any other organ. Percussion generally betrays the presence of the intestine filled with air in front of the tumor. At the same time the tumor sometimes compresses the colon, its walls are pressed together, and percussion gives a dull resonance. But, by careful palpation, through relaxed abdominal walls, we may sometimes succeed even then in feeling the empty, compressed, descending colon as a cylindrical band between the tumor and the abdominal walls. In some isolated cases the colon, running down over the tumor, may even be recognized by inspection.

In a case of Faludi's,[1] in a boy five years old, it could readily be observed, especially at the beginning of the disease, that a longitudinal strip ran down over the tumor, apparently dividing it into two parts. This sometimes disappeared and then again reappeared, according as the descending colon, passing down over the surface of the tumor, was filled with fæcal matter or gas, or collapsed after they had passed away.

The tumor *does not* follow the movements of the diaphragm, and is generally immovable. Cancerous kidneys, in spite of their great size and weight, are comparatively seldom movable, probably on account of the adhesions which the degenerated organ forms with the surrounding parts. Rollet speaks of a movable cancerous kidney, and in the Lancet of the 18th of March, 1865, a cancerous wandering kidney of the right side is reported, which was taken for an ovarian tumor. On palpation these renal tumors almost always show a certain degree of elasticity. Sometimes they are round and smooth, sometimes nodular and irregularly lobulated. Individual nodules often present various degrees of hardness. Occasionally individual portions of the tumor, or even the whole of it, may convey a simulated sense of fluctuation. There are tumors caused by renal carcinoma, in which auscultation reveals a blowing murmur. Ballard[1] reports such a case, and states that the murmur was so loud that Bright felt obliged to diagnosticate an aneurism of the renal artery. Bristowe[2] observed an analogous case.

---

[1] Jahrbb. f. Kinderheilk. VII. 1865.

The second most important symptom is *hæmaturia.* It is more rarely observed than the tumor. While the latter was only absent three times in fifty-two cases collected by me, hæmaturia occurred only twenty-four times in fifty cases. It is, furthermore, no pathognomonic sign of renal cancer ; for it is to be observed in many diseases of the urinary organs, and especially of the kidneys. But it is none the less a very important sign where it is present, inasmuch as by its frequently early appearance it attracts the attention of the physician. Hæmaturia, as has been remarked, often appears far earlier than the tumor. Not infrequently it appears at the very beginning of the disease, not recurring afterwards until death. Long intervals may elapse between this initial hemorrhage and the occurrence of other symptoms.

In some cases of renal carcinoma such hæmaturia was followed, some time afterward, by the passage of a renal calculus, which seemed to account for it. In other cases the renal hemorrhage lasts for a longer time, and frequently returns. It recurs at irregular intervals of weeks or months, or of a few days. In some instances renal hemorrhage does not occur till shortly before death ; indeed, in a case obesrved by Gairdner, it did not appear till the day of death. It generally takes place without any external violence that could be considered as causing it. At the same time it cannot be denied that a fall, or a blow, or some other traumatic injury, especially one affecting the region of the loins, does often precede it. Brinton[1] describes a case in which a hemorrhage from the urinary passages following an injury was the only symptom of a renal cancer. In these traumatic cases the hæmaturia is generally very profuse. Renal hemorrhages in carcinoma are never so scanty as to require the use of the microscope for their detection. The blood-corpuscles are sometimes intact, sometimes altered, while tube-casts covered with blood-corpuscles are often found. The blood is generally intimately mixed with the urine, which varies in color according to the amount of blood. Sometimes it is more or less red, sometimes blackish. Even in children renal hemorrhage is seldom excessive or exhausting. Sometimes the urine contains a greater or less number of larger fibrinous clots, the blood becoming coagulated in the pelvis of the kidney or the ureter, and afterward being floated away with the urine.

---

[1] Transactions of the Path. Soc. 1859.

[2] Med. Times. 1854. II. p. 395.

The hemorrhages themselves take place without any suffering, and are hereby to be distinguished from those that will be described in connection with renal calculus. But the escape of clots, as we shall soon see, is often associated with great suffering. Occasionally the hemorrhage ceases suddenly. This often depends on an obstruction of the ureter, which may be occasioned by blood-clots or by cancerous masses. The closure of the ureter may also be caused by compression on the part of the renal tumor. On closure of the ureter of the diseased side, urine is excreted by the healthy kidney alone. Hæmaturia is sometimes followed by complete anuria when, for instance, the neck of the bladder or the urethra is closed by a clot.

The reaction of the urine in renal cancer presents nothing specially worthy of note. The amount of urine is almost always normal, in some cases it is more abundant than natural. Occasionally a slight diminution in amount has been observed. In hæmaturia the presence of fibrin and albumen can be demonstrated. Albuminuria without hæmaturia is rare, except when pyuria or nephritis are present at the same time. Uræmic symptoms are very rarely observed. White blood-corpuscles, epithelial cells, and detritus from the tumor have been found in the urine. If renal cancer is complicated with suppurative processes within the urinary organs—as, for instance, in rare cases, with pyelitis—then a purulent sediment is found. In one case (Jerzykowsky), under heavy pressure, urine was frequently evacuated containing a peculiar mucilaginous substance. Unfortunately, this was not more accurately examined.

---

[1] British Med. Journ. 1857.

The presence of cancerous masses in the urine, which is often alluded to as a symptom of renal cancer,[1] seems at most to be but rarely observed, although the physician may have directed his entire attention to this important point. Heller states that in some few instances of renal cancer he has found deposits of uric acid as well upon *discharged flocculi of cancer tissue* as upon the waste substances obtained at the autopsy. In many other instances adduced in proof of this, there is no doubt that the epithelium of the renal pelvis and the ureters has been mistaken for cancer-cells. This certainly also holds true with regard to the observation of Moore,[2] so often cited. He even took the urine from the bladder after death, and thought he could diagnosticate a cancer from the accumulation of roundish and caudate cells, etc. The discovery of certain cell-forms in the urine is of no sort of value in the diagnosis of cancer; the only thing that can be regarded as a significant symptom is the discovery of *cancer-particles with an alveolar structure.*

Quantitative changes in the composition of the urine have thus far been but little met with in this disease. Doederlein, in his case, found the metamorphosis of the nitrogenous elements of the body to be strikingly great and increased, notwithstanding that, during the latter part of the time, the temperature of the body was lowered, respiration slow, and the amount of nourishment taken very slight.

In renal cancer, pain is a symptom of very variable significance. It is sometimes frequent, sometimes rare; now intense, then again insignificant; it may be very agonizing, but it may also be entirely wanting, even in large tumors. The absence of pain is not enough to exclude the idea of the existence of a renal cancer.

---

[1] Thus, even in Johnson's Renal Diseases, German edition, p. 388.

[2] Medico-Chirurg. Transact. XXXV. Case of a pulsating tumor, in which the urine contained cancer-cells.

Pain may occur in severe paroxysms, or it may be dull, deep, and continuous. Most frequently it involves the entire region of the loins and the hypochondrium of the diseased side. Sometimes patients complain of a sense of compression on the affected side. Tolerably often the pain extends along the last intercostal spaces, sometimes it radiates through the lower extremities, so that disease of the hip is suspected. As long as no tumor can be felt and there is no hæmaturia, the sciatica is sometimes thought to be rheumatic. The pains, which are generally caused by compression of the sciatic nerve through carcinomatous lymphatic glands, become aggravated to an unbearable degree. Serious disturbances of sensation arise, followed by emaciation of the extremity affected. Sometimes the tumor cannot be detected until this advanced stage is reached. In some cases the pain arises spontaneously, in others it only follows on pressure exercised over the diseased parts.

Retraction of the testicle, such as is common in nephrolithiasis, is seldom met with in renal cancer. The pains may simulate those of renal calculus, which, indeed, is by no means so rare a concomitant of cancer. Much more frequently, however, these pains result from the passage of fibrinous clots, which are not uncommon after renal hemorrhage. Sometimes these become wedged into the lower, narrow extremity of the ureter, and severe colic pains result.

Renal cancer is occasionally accompanied by disturbances of digestion, which may arise quite early. The appetite fails; nausea and vomiting occur from time to time. In other cases these signs are altogether wanting; in some a voracious appetite has even been observed, especially in children, occasionally accompanied with increased thirst. As a rule, constipation persists during the greater period of the disease; during the more advanced periods it often alternates with diarrhœa, and sometimes continuous diarrhœa exists. Now and then, in cancer of the right kidney, icterus has been observed as the result of compression of the ductus choledochus.

The condition of the patient's strength varies very much according to the stage of the disease. In adults, notwithstanding the presence of a tumor, a year or more may elapse before the general well-being is affected, but in many patients there is an early loss of strength and a cachectic appearance.

The pulse shows nothing striking, though occasionally it is perceptibly retarded. There is no fever; the temperature has sometimes been observed to be subnormal. Towards the close of life febrile complications are occasionally encountered. In large, rapidly growing tumors respiration is often early interfered with if the tumor compresses the lungs and hinders the movements of the diaphragm. Œdema often occurs, sometimes restricted especially to the lower extremity, as the result of thrombosis of the inferior vena cava, sometimes in the domain of other large abdominal veins. For the same reason the cutaneous veins of the abdomen seem enlarged. These run a tortuous course as a largely dilated venous network, in extreme cases being as thick as a quill, and by abundant anastomosis facilitate the return of venous blood. Now and then general œdema is developed as the result of increasing anæmia and hydræmia. The mental powers usually remain intact until the fatal termination, which most frequently follows through exhaustion.

Complications.

These are generally caused by secondary cancerous developments. At the same time they are rare. Thus secondary cancer of the lungs gives scarcely a symptom. Secondary cancer nodules of the liver sometimes become so large that they can be felt. Secondary cancer of bone-tissue manifests itself by very severe pain; if the cancer spreads to the vertebræ it may result in evidences of compression of the spinal cord, painful paraplegia, etc.

Cornil[1] has reported such a case. In a woman, thirty-three years of age, cancerous degeneration of the left kidney had resulted in an extension of the process to the last two lumbar vertebræ, involving the dura mater, and in this way causing compression of the nerves of the cauda equina. The result of this secondary deposit was motor paralysis of both legs, with almost complete anæsthesia and a loss of reflex irritability in the same. Aside from this, death may also result from other complications, serious hemorrhages, or peritonitis, which is sometimes brought about by rupture of the cancer. In one case of Bright's, death followed rupture of the carcinoma and fatal hemorrhage into the abdominal cavity.

---

[1] Mém. de l'acad. de méd. XXX. p. 387.

Diagnosis.

The diagnosis of renal cancer is almost always difficult and often quite impossible. He who has the opportunity of seeing the patient during the entire course of the disease, noting the history of its development and its course, the successive appearance of hæmaturia and a tumor of the kidney, etc., will find it more easy to recognize the seat and nature of the tumor. Familiarity with the experience of others is of value in making a diagnosis. Large renal tumors in young children, for instance, are hardly ever caused by anything else than cystic degeneration, genuine hydronephrosis, and especially primary medullary carcinoma. As the two first no doubt invariably appear as congenital affections, the diagnosis of medullary carcinoma may be made, by exclusion, with a degree of probability bordering on certainty in case of non-congenital renal tumors in children. But if the main symptoms just described—tumor and hæmaturia—are absent, then it is generally impossible to make even an approximately probable diagnosis; for pain in the region of the kidneys, even when it is present, occurs in a whole series of kidney affections, and it possesses no characteristic distinguishing peculiarities. If, however, the kidney pain is continuously severe, if cancer unmistakably exists in other organs, if this trouble was preceded by the sloughing of cancerous testicles, then the pain alone argues in favor of the extension of cancer to the kidneys, even if other symptoms fail. If an abdominal tumor is found in a patient, associated, at the same time, with hæmaturia, we cannot but think at once, among other possibilities, of the chance of cancer of the kidney. Cases in which there is merely swelling of the kidney may escape the diagnosis of most excellent observers, inasmuch as they run quite a latent course. Thus, Lebert reports a case[1] in which, on opening the body of a woman who had died of pleuritis and pericarditis, he found a carcinoma of the kidney nearly as large as his fist, which had run an entirely latent course. If a tumor is found, the next question to be answered naturally is whether it actually belongs to the kidney. This is often no easy task. Even Bright asserts that among abdominal tumors few are so difficult to recognize as tumors of the kidney. Enlargements of the kidney, according to all experience, are very often confounded with tumors of other abdominal organs. Those of the right kidney may be mistaken for glandular tumors of the portal fissure, for tumors of the liver, for disease of the pylorus, of the cæcum, or of the ascending colon ; those of the left kidney for tumors of the spleen and diseased conditions of the descending colon ; those of both kidneys for tumors of the mesenteric glands, the ovaries, and the uterus.

Attention may be called to the following clinical points in differential diagnosis.

Renal tumors are to be distinguished from *enlargements of the liver* by the fact that the former do not rise high into the thorax, and likewise do not crowd the liver far upward. When the patient lies on his back, it is generally practicable to pass the hand between the edge of the ribs and the tumor. This is not the case in a tumor of the liver. Bright himself called attention to this important distinguishing sign. The only kind of liver tumor that might here lead to error would be one that arose from the posterior portion of the organ, and was isolated. But they probably never separate themselves so far from the liver structure in which they are imbedded as to be mistaken for renal tumors. Furthermore, if no displacement of the intestines has taken place, the ascending colon almost always lies in front of the renal tumor, passing obliquely from below and to the right upward and to the left. This probably never takes place with the liver, unless it be in an exceptional case of malformation and atrophy. Tympanitic resonance on percussion, or the passage of wind through it, is generally enough to determine the position of the colon. If tumors of the liver and kidney complicate one another, which is sometimes the case, especially in malignant neoplasms, it may be quite impossible, in a diagnostic point of view, to rise above the realm of conjecture. But if the urine gives some sign (hæmaturia), and one can feel hard knots in the liver, then the diagnosis may, after all, be made with some degree of probability.

---

[1] Virchow's Archiv. XIII. p. 532.

Distention of the cæcum and ascending colon, through fæces and gas, which has given rise to so many diagnostic errors, has also been mistaken for a tumor of the kidney. Still, as a rule, a sufficient number of fixed points for the diagnosis are furnished by the fact that in fæcal accumulations percussion gives a dull sound at one point and a tympanitic sound at another, which, added to the doughy feel of the tumor, the fact that its form can be changed by kneading, a careful consideration of the history of the case, and, above all, the effects of a cathartic, should be sufficient to prevent error.

If the tumor is situated on the left side, great difficulty may be experienced in determining whether it is of the kidney or spleen—a question which was ventilated by Troja. The renal tumor extends deeper down, and not so far up as that of the spleen. The anterior margin of an enlarged spleen is generally very easily and distinctly to be felt. It is also usually easy to pass one's finger to the lower surface of the splenic tumor and to feel the indentations which are often present in this organ. Special diagnostic value is furthermore attached to the fact that in renal tumors the descending colon lies in front of the tumor, which is not the case in tumors of the spleen. Spencer Wells has recently advised the injection of air per rectum in cases in which one does not know whether a loop of intestine passes over the tumor, for the purpose of distending the collapsed intestine and facilitating the diagnosis. At the same time there are also many exceptions to this position of the gut. Rosenstein reports a case of left-sided renal carcinoma in a boy, in which the dullness communicated by the spleen passed directly into the dullness caused by the renal tumor. On post-mortem examination the descending colon was found pushed entirely to the rear, and pressed flat. Renal cancers are now and then confounded with tumors of the spleen. Quite recently Gjoer observed a case of a carcinoma of the left kidney as large as a man's fist, in which hæmaturia and the tumor were both present, and yet the latter had been taken for an enlarged spleen. But a large abdominal tumor on the left side, associated with hæmaturia, should not mislead one into making the positive diagnosis of renal cancer, for profuse hæmaturia sometimes occurs in leukhæmic patients. Roberts relates a case of an enormous leukhæmic tumor of the spleen, in which a profuse hæmaturia was observed, lasting several days. Nevertheless, at death, which followed in a few months, the kidneys and bladder were found healthy. This case might have been cleared up by an examination of the blood.

Ovarian tumors are not as often confounded with renal cancer as with other tumors of the kidney. Still, such diagnostic errors do occur when the tumor develops itself forward against the navel, the pubic and iliac regions, as may be the case in a cancerous wandering kidney (compare Diagnosis of Wandering Kidney). The development of the tumor from the cavity of the true pelvis here naturally argues in favor of an ovarian tumor (unless, perchance, a kidney, located in one of the cavities of the true pelvis, undergoes cancerous degeneration). But, as this circumstance can generally only be determined by the history of the case, and as with most patients this is not reliable enough, we must in the majority of cases avail ourselves of other diagnostic points. There are scarcely ever any loops of intestine to be found in front of an ovarian tumor; they are usually crowded away to the lateral regions of the loin, where they give loud resonance on percussion, just in the region, therefore, where there should be the greatest dullness if the tumor proceeded from the kidney. This symptom also holds good when it is a question of distinguishing between a renal and a uterine tumor. Aside from this, of course, certain points are determined by internal exploration, and, finally, by the complete rectal examination after Simon's method. Here, too, however, the differential diagnosis offers great difficulties. Greenhalgh[1] observed what was supposed to be an ovarian tumor, as a complication, during two pregnancies. The chances for ovariotomy were being debated when the woman became pregnant a third time. She died before her confinement, without any very clear cause of death. The supposed ovarian tumor was a medullary cancer of the kidney in an advanced stage. Such cases should be cleared up by an exploratory puncture before any serious operative interference is undertaken.

---

[1] St. Bartholomew's Hosp. Rep. Vol. I.

In some rare instances these cancers have been mistaken for *aneurisms*, especially where on auscultation and palpation whirring sounds could be heard and felt in the tumor. They have also been mistaken for *ascites*, when the cancer was very soft and filled the entire abdomen. Such a case, occurring in a girl three years old, is reported in St. George's Hospital Reports, II. In ascites, however, both lateral regions of the abdomen are dull, while here the dullness is confined to one side.

It is very much more common to confound a *psoas abscess* with renal cancer, especially during the age of childhood. Aside from the earliest stages, where a differential diagnosis is impossible, it is very difficult to determine the matter even after the formation of a tumor. In both cases the tumor occupies the region of the loins and extends forward into the abdomen. It is often difficult or even impossible to feel the fluctuation in the abscess, and the diagnosis is still further complicated by the fact that large, soft cancers also give a distinct feeling of fluctuation. At the same time, psoas abscess never extends as high up in the abdomen as renal abscess, and the sensitiveness of psoas abscess is far greater than that of renal carcinoma.

In children we must also bear in mind the *immense bundles of cheesy glands* that are sometimes present in the abdomen, and which, when symmetrically situated in both hypochondriac regions, may now and then be confounded with renal tumors.

Being once satisfied that a tumor proceeds from the kidney, the next point is to determine its nature. The following signs argue in favor of carcinoma: *rapid and irregular growth of the tumor*, *inequality and nodular character of its surface*, *its varying consistency at different points*, being harder in one spot and softer in another. In general, such a tumor has a firmer consistency than cystic tumors containing fluid. Occasional attacks of hæmaturia aid the diagnosis, which is strongly confirmed if secondary cancer nodules can be distinctly felt in other organs. Among the individual diseases of the kidney which lead to an increase in volume, and which must be considered in making a diagnosis, we may mention hydatids of the kidney, renal cysts, suppurative pyelonephritis, suppurative perinephritis, and hydronephrosis. The points essential to a differential diagnosis are treated of in connection with each one of these diseases. If two or more of these affections are combined in any given case, as not infrequently happens with regard to nephrolithiasis and carcinoma, then the diagnosis is often very difficult. With regard to the differential diagnosis between sarcoma and carcinoma of the kidney, compare page 636.

An *exploratory puncture* is one of the aids to diagnosis which has but seldom been used in practice. It was employed in Doederlein's case. A whitish red mass of tissue was obtained, similar to the medullary matter of the brain, in which the microscope showed a delicate, connective tissue stroma, in which innumerable nuclei were embedded. This settled the diagnosis of the character of the tumor. The operation was followed by no bad results. Schueppel[1] reports an observation made on a man forty years of age, who, in addition to great ascites, had a colossal tumor in the right hypochondrium. A hydatid tumor was suspected. On making an exploratory puncture, little gelatinous bodies were discharged of the size of a grain of pepper up to that of a pea, in which there were no hooks nor membranes, but only a homogeneous, mucilaginous mass with isolated cells that had undergone granular and fatty degeneration. We are not informed whether the puncture did any harm. Carcinomatous tumors of the kidney, which were taken for abscesses, have been punctured by mistake.[2]

Duration, Course, Prognosis.

It is hard to estimate its duration, because the beginning of the disease cannot be fixed with certainty, but, at most, only approximatively. This much may be said in general: that renal cancers run their course more rapidly during the age of childhood —at which time they proliferate more quickly—than at a more advanced age. In delicate children their duration is often to be counted by weeks. In one instance the period during which the objective symptoms could be observed was five weeks. As a rule, they last about nine months, hardly ever more than a year to a year and a half. I know of but one case that lasted two years. In adults, however, the disease may extend over several years, generally not over three or four, though here, too, the fatal termination is sometimes reached within a year. In but one case was the disease demonstrated to have lasted eighteen years.

---

[1] *Eberhard's* Dissertation, p. 17.

[2] *Barth*, Bullet. de l'Acad. XXXV. Nov. 1870.

The termination is inevitably fatal, the prognosis bad. In some cases the disease seems to come to a standstill, an apparent improvement taking place. Brinton states that in his case, under the vigorous internal and external use of preparations of iodine, a striking improvement took place. Death followed suddenly in this patient by hemorrhage into the tissue of the tumor.

Therapeutics.

Here medical science celebrates no triumphs. The only thing to be done is to maintain the strength of the patient, so far as possible, by good regimen, to quiet pain by narcotics where this is practicable, and to combat dangerous symptoms, such as hæmaturia. During these attacks of renal hemorrhage, absolute rest in bed, ice-bladders to the abdomen, over the tumor, the internal use of acetate of lead, tannin, alum, and ergotine, are the best measures that one can recommend. If the flow of urine stops after such hemorrhages, we must see whether the urethra is filled with clots; if so, they are to be pushed back into the bladder with a catheter, and afterwards removed so far as possible by injections of lukewarm water. The removal of cancerous kidneys, which has been undertaken a few times as the result of errors of diagnosis, does not fall within the range of therapeutic effort. The most successful result attained was by Wolcott.[1]

---

[1] Phila. Med. and Surg. Rep. 1861. p. 126.

He extirpated a cancerous kidney which he had taken for a hepatic cyst. The tumor weighed about two and a half pounds. The patient survived the operation two weeks.

Among *lymphatic* neoplasms, the first that are worthy of interest are those originally described by Virchow,[1] and afterward by Friedreich, Boettger, and others, in connection with leukhæmia, and which were furthermore also described by E. Wagner,[2] and subsequently more accurately by C. E. Hoffmann in connection with typhoid fever. These lymphatic neoplasms appear partly in a circumscribed and partly in a diffuse form. At first a deposit of lymph-cells takes place around the Malpighian bodies. These, like the uriniferous tubules, are compressed by the growing neoplasm. At the same time they can still be demonstrated, lying within the neoplasm in an atrophied condition. These growths are to be distinguished from tubercles by the absence of caseation. Under this head we must likewise consider

Tubercles of the Kidney.

Literature.

Beer, Die Bindesubstanz der menschlichen Niere. 1859. p. 187.—*Virchow*, Geschwülste. II. p. 654.—*Wilh. Mueller*, Structur und Entwickelung der Tuberkeln in den Nieren. 1857.—*E. Wagner*, Archiv f. Heilkunde. XII. p. 10 and 12.—*The same*, Tagebl. d. Leipziger Naturforscherversammlung. 1872. p. 214. —*Cornil*, Arch. de phys. normale et pathol. I. (1868.) p. 105.

Pathology.

Tuberculosis of the kidney is commonly one of the manifestations of general miliary tuberculosis, in which miliary tubercles are developed in the kidneys, as well as in other organs, and are often surrounded by a highly injected areola. The superficial tubercles, or those lying in the convoluted tubes of the cortical substance, are round in form ; those lying between the straight tubes of the medullary and the cortical portions are more elongated and linear in form. The epithelial cells of the kidney are generally in a state of advanced granular degeneration. This participation of the kidneys in the tubercular process generally causes no change in the group of symptoms of acute miliary tuberculosis. It is, therefore, of no clinical interest.

---

[1] Arch. V. Ges. Abh. p. 208.

[2] Virchow's Archiv. 1860. p. 325.

There used to be, and is even now, a widespread impression that the miliary renal tubercles just described, by forming into groups—which is certainly often observed—also amalgamate into large nodules and infiltrations, which show a caseous character and afterward break down and form cavities, and then present the picture of renal phthisis. This form has been described as primary tuberculosis. In the description heretofore given of renal phthisis (p. 577), I separated it from tuberculosis, because it is very often to be regarded as the result of a chronic inflammation with cheesy metamorphosis, and not as the result of caseating confluent deposits of tubercle. It has already been intimated, in the account of renal phthisis, that the two processes often enough take place simultaneously. But then the tubercles are something accidental, just as they may also be developed, for instance, in the vicinity of caseous inflammatory deposits within the lung. But just as, to-day, we no longer consider this as belonging to tuberculosis, that is, as resulting from the confluence of miliary tubercles in the lungs, so it seems unreasonable to admit the doctrine as applied to analogous processes in the kidney, the renal pelvis, and the ureter.

Foreign Bodies in the Kidney, the Pelvis of the Kidney, and the Ureter.

Nephrolithiasis.

(Renal Gravel, Renal Calculus, Calculi Renum.)

History and Literature.

From the earliest periods, renal calculi and the symptoms which they produce have attracted the attention of physicians, and, in many respects, their descriptions of the same are tolerably complete. With the development of the study of pathological anatomy, since the time of Morgagni, the alterations produced by renal calculi in the kidney, its pelvis, and the ureter, have been more carefully studied. The chemical investigation of renal stones remained for a long time a *terra incognita.* Physicians and chemists, from Galen to Paracelsus, from Paracelsus to Van Helmont and Boerhave, by their vacillating and often incomprehensible reasoning on this subject, furnished no material of any value touching on the doctrine of the composition of renal stones. It was only by the great discovery of the distinguished Swedish chemist Scheele, in 1776, that uric acid was recognized both as an ingredient of renal calculi and as a normal constituent of the urine. It is true that Scheele's investigations were restricted by the belief that all renal calculi consisted of uric acid. After Scheele's discovery, the labors of Fourcroy and Vauquelin, in France, and Wollaston, the discoverer of cystin, in England, as well as the detection of phosphates in the calculi, contributed most largely towards raising our knowledge of renal calculi to its present position. These labors were worthily followed up by Marcet, Berzelius, and a number of other investigators. Thereby urinary concretions, which had been judged solely by their external appearance, were transferred to the domain of chemistry, which almost entirely mastered them. By this means the subject of lithontriptic remedies, although it had been agitated and discussed from the earliest times, was placed upon a different basis, inasmuch as the concretions, formed of definite components, could be opposed by definite solvents. These therapeutic questions excited discussions, which were conducted with great acrimony, as, for instance, by Civiale, but which had the tendency to restrain within proper limits many extravagances by which the entire method was in danger of being thrown into disrepute.

The older literature of the subject may be found collected in the sixth volume of Naumann's Handbuch der medicinischen Klinik, Berlin, 1836, and Canstatt's Handbuch der medicinischen Klinik, Vol. IV. 3d division, 2d edition, Erlangen, 1845. In the account here given the general literature collected on page 543 was made use of, as well as the following works:

Joh. Varandaeus, Tractatus de affectibus renum. Hanoviae, 1617.—*Boerhave* de calculo. Londini, 1741.—*Sydenham's* Works, Vol. II. London, 1788.—*Fr. Aug. Walter*, Einige Krankheiten der Niere und Harnblase. Berlin, 1800.—*Joh. Peter Frank* (1810), Spec. Pathol. und Therapie. Deutsch von *Sobernheim*. II. Bd. S. 440. 1840.—*Marcet*, An Essay on the Chemical History and Medical Treatment of Calculous Disorders. London, 1819.—*Magendie*, Recherches, etc. sur les causes etc. de la gravelle. Paris, 1827.—*Brodie*, Lectures on the Urinary Organs. London, 1832.—*Civiale*, On the Medical and Prophylactic Treatment of Stone and Gravel. Translated by H. H. Smith, M.D. Philadelphia, 1841.—*Willis*, Urinary Diseases and their Treatment. Philadelphia, 1839.—*Bence Jones*, On Gravel, Stone, and Gout.—*Prout*, On the Nature and Treatment of Stomach and Urinary Diseases; being an Inquiry into the Connection of Diabetes, Calculus, and other Affections of the Kidney and Bladder, with Indigestion. London, 1843.—*Schlossberger*, Archiv f. phys. Heilkde. 1850.—*Hodann* u. *Mueller*, Günsburg's Zeitschrift f. klin. Medicin. II. (1851) S. 264.—*Rilliet* et *Barthez*, Traité des maladies des enfants. 1853. 2me éd. T. II. p. 38.—*Hodann*, Verhandlungen d. schles. Gesellsch. u. s. w. 1855.—*Virchow*, Gesamm. Abhandlungen. 1856. S. 833.—*Meckel*, Mikrogeologie. 1856.—*Todd*, Clinical lectures etc. on the the urinary diseases. 1857.—*Heller*, Die Harnconcretionen 1860.—*Garrod*, The Nature and Treatment of Gout and Rheumatic Gout. London, 1859.—Virchow's Archiv Bd. X. S. 230; Bd. XI. S. 217; Bd. XXVI. S. 419.—*Hirsch*, Handbuch der historisch-geographischen Pathologie. II. S. 349. 1862.—*Owen Rees*, Guys hospit. Reports. 1864. p. 214.—*Thompson*, Clinical lectures on the Dis. of the urinary organs. London, 1868.—*Julius Mueller*, Archiv der Pharmacie. 1872. 51. Jahrg. S. 308.—*Braun*, Balneotherapie. 3. Aufl.—*G. Simon*, Verhandlungen der deutschen Gesellschaft für Chirurgie. II. 1873. Berlin, 1874.—*Beneke*, Grundlinien der Pathologie des Stoffwechsels. Berlin, 1874.

Etiology.

Renal gravel and larger renal concretions consist of and are developed from both normal and abnormal ingredients of the urine. In order clearly to understand the method of production of larger concretions, we naturally begin by studying the secretion of amorphous renal gravel—that is, the powdery sediments which are evacuated with the urine. They constitute the starting-point and the basis of what is ordinarily called renal gravel and renal stones.

Formerly, men were satisfied to account for the production of renal gravel on the theory of the existence of certain diatheses—and indeed, to some extent this is true even now. They thought that the stone-producing substances were either abnormally produced in the kidney, as, for instance, cystin, or were secreted and thrown down in excess, as for instance, uric acid, whereupon, then, under certain circumstances, larger concretions were formed out of them. According to the principal classes of renal concretions they then distinguished a uric acid diathesis, an oxalic acid diathesis, and a phosphatic diathesis. Although this view comprises a certain element of truth, yet, expressed in these general terms, it is false. All these concretions, for instance, may be developed without the presence of any diathesis whatever. The attempt to establish a *phosphatic diathesis* places one in the worst predicament, for the phosphates cease to be held in solution as soon as the urine becomes alkaline, as the result of an inflammation or a local catarrh of the urinary passages, without the formation or presence of any additional amount of phosphates. As regards the so-called *uric acid diathesis*, it would be quite incorrect to assert that uric acid concretions are always developed from constitutional causes. The evidences that were formerly adduced for this were false, for the excretion of uric acid precipitates never justifies the conclusion, without further proof, of the presence of an increased amount of uric acid. It is quite correct that the formation of an increased amount of uric acid in the body may and does become the occasion of the excretion of the same through the kidney ; and yet, in the presence of a normal or even a diminished amount of uric acid, the same effects may be produced, supposing that conditions arise under which the normal or even diminished amount of uric acid cannot be held in solution. Among the conditions that will have this effect are increased activity of the urine, a diminished temperature, which was assigned by Magendie and Ségalas as the cause of the predisposition of old people to urinary calculi, as well as the presence of solid bodies in the urine, which greatly favor the release of the solid constituents of the urine from the solution in which they are held. Increased acidity of the urine may be brought about in various ways. It may result from the presence of an increased amount of certain constituents of the urine (acid biphosphate of soda) which withdraw the soda from its combination with the uric acid, by means of which combination it is held in solution as urate of soda. It may also result from acid fermentation of the urine,[1] which, it is true, does not generally take place till after the evacuation of the urine, though sometimes it does arise within the urinary passages. In that case the amount of acid is probably increased by a decomposition of the coloring and extractive matters of the urine. The mucus of the urinary passages seems to act as the leaven that induces fermentation.

Passing to the so-called *oxalic acid diathesis*, we find that oxalic acid may, under pathological conditions, exist in the animal body as the result of disintegration. But it may also result from the food introduced. Schultzen[2] claims to have found it in the urine, in minimum quantities, even under normal conditions. On account of its great affinity for lime, it unites with the same to form oxalate of lime, which is very easily recognizable under the microscope by its pretty octohedra (marked like the open side of an envelope). In view of its great insolubility, this is very apt to give rise to the formation of gravelly deposits. Cystin appears to arise, in the human body, only under pathological conditions, which seldom occur and of which nothing further is known than that they are sometimes hereditary. In acid urine, cystin is at once crystallized, and is then discharged as gravel.

---

[1] Compare *Scherer*, Untersuchungen zur Pathologie. 1843.

[2] Reichert u. Dubois' Archiv. 1868. p. 719.

With regard to the solid bodies which are so liable to serve as the nucleus for crystalline deposits, the probabilities are that, in this country, the only process that plays any figure in the matter is the increased secretion of mucus in the pelvis of the kidney. It may be that, in the kidneys themselves, in rare instances, tube-casts serve as points of deposit of crystalline formations, especially of oxalic acid. In Egypt, Griesinger saw the eggs of the *distoma hæmatobium* forming the nucleus of renal calculi. There are no positive observations in support of the view, which has been widely accepted, that blood-clots constitute the starting-point of renal concretions. Meckel considered the formation of these concretions to depend on the existence of a peculiar mucous catarrh, and regarded oxalate of lime as the active agent in the petrifaction of the mucus produced within the pelvis of the kidney. Notwithstanding the ingenuity of this and of other theories laid down in his Microgeology of Renal Stones—such as their double growth, partly by the apposition of new layers from without, partly by intussusception, the theory of the continual metamorphosis of matter taking place within them, of the trans-substantiation of stones, etc.—they have not commanded general recognition, because they are not founded on facts.

We know very little about the *predisposing causes* of renal calculus, for we are not yet in possession of any accurate statistics with regard to stone in the kidney proper. All that we have is some scattered material with regard to the statistics of urinary calculi in general, which material August Hirsch has collected, sifted, and criticised in his classical work on historical and geographic pathology (1862, Vol. II. p. 348). Even if the greater part of the urinary calculi originate in the kidney (according to Heller's experience the proportion is as 100 to 1), still the results of this investigation may only with great reserve be applied to renal stones.

With regard to the *age* at which renal stones are observed, they occur with special frequency during the period of childhood—a fact which rarely holds good with reference to gallstones. Concretions in the kidney may even be found during fœtal life, a number of instances of which are given in the older literature of the subject.[1] Concretions of various sizes are found tolerably often in the renal pelves and calyces of young children, especially those that have attained the age of a few months. They are almost always little oxalic acid stones. J. P. Frank mentions two cases, one in a child two days old, and the other in one eight days old, in which death occurred by convulsions during the passage of little calculi. The period of life most subject to the disease is that of childhood before the age of five; the next most obnoxious period is that from five to fifteen. Among 5,900 calculi, which were observed in various places, Civiale found 45 per cent. to have occurred in children. During the period of youth and maturity the tendency to the formation of calculus diminishes, to return again in later years.

It has been ascertained that the children suffering from vesical calculus ordinarily belong to the lower classes in society, while the adults suffering from the disease belong to the better classes. We have as yet no experiences based on a sufficient amount of material to justify us in judging how far the same rule may apply to renal concretions.

The *male sex* is by far the most liable to urinary calculi. Over against 5,497 cases occurring in males we find only 309 in females.[2] This holds good of adults as well as of children. Among eight children in hospital affected with stone, Barthez and Rilliet found six to be boys and two girls. The same relation was maintained in their private practice. A similar experience was recently reported by Neupauer,[3] who, in one hundred cases, found but five girls. A circumstance not to be underestimated in calculous disease is that of *heredity* and *family idiosyncrasy*. This is especially striking in cystin stones. But it is by no means constant. It was formerly thought that *certain regions enjoyed immunity from calculous disease*. It was said, for instance, that the tropics were free from such affections. This is a mistake. Allan Webb[4] also receded from his view that stone in the bladder was an unknown disease in India, when he found in a museum about 300 specimens of stones, originating in various patients who had been operated on in all parts of India. Heinemann[1] has also recently communicated the information that renal calculi and the conditions following them are tolerably frequent in Vera Cruz. We know now that some parts of the tropical and sub-tropical regions are quite prominently visited with this affection. This is very easily to be understood, on physiological grounds, because the more abundant the activity of the skin the more concentrated will be the urine, and because the liability to urinary deposits, and therefore to gravel and stone, stands in direct relation to the proportion of solid ingredients in the urine, especially to the amount of uric acid it contains. Accordingly, the former view that a damp, cold climate was an essential cause for the development of calculous disease, must be regarded as entirely without foundation. Certainly the endemic occurrence of this disease in the east of England and in the Netherlands cannot be referred to climatic influences. For, so far as England is concerned, it has worse climates than that of its eastern portions, and in the Netherlands calculous disease has materially diminished since the beginning of this century, without any change of climate being observed. *Habits of life* and *conditions of nutrition* have undoubtedly a prominent influence in the development of renal stones. Although the statements of former observers—of Boerhave, for instance—to the effect that renal calculi might be developed merely as the result of prolonged lying still, do not seem to have been founded on sufficient and reliable observation, still the prevalence of this disease mainly among aged persons in the higher walks of life, who indulge in a quiet, sluggish method of life, subsisting chiefly on a meat diet, would go to prove that bodily inactivity, together with a rich, nitrogenous diet, constitutes at least one factor in the etiology of urinary calculi. On the other hand, the fact that among children it is mainly those of the poorer classes who are attacked, has been explained on the ground of the coarse, indigestible, principally amylaceous food with which they are supplied. The endemic occurrence of urinary calculi in some regions is probably far more referable to some such faults in the conditions of nutrition than to unfavorable climatic peculiarities of the regions concerned. At the same time, the importance of these circumstances is not to be overestimated, for, after all, the number of instances in which this disease occurs is very small compared with the extent and the uniform distribution of the faults of nutrition above referred to; and, moreover, the disease is often not to be found at all in regions where these faults exist in the most pronounced manner. The same may be said with regard to the causative influence of the use of sour young wine, and various other things which have been credited with producing stone in the bladder. At all events, the specific etiological cause is as yet unknown, which admits of innumerable cases of the abundant and continued formation of gravel without the formation of a stone. As long as this question is not solved, we are deprived of one of the principal elements in the pathogenesis of urinary concretions.

---

[1] Compare *Graetzer*, Krankheiten des Fötus, 1837, as well as *Naumann*, Handbuch der medic. Klinik. Bd. VI. 1836. p. 462.

[2] *Oesterlen*, Handb. d. medic. Statistik. 1865. p. 649.

[3] Jahrb. der Kinderheilk. V. Heft. 4. 1872.

[4] Pathol. indica. p. 245. Calcutta. 1848.

---

[1] Virchow's Archiv. LVIII. p. 183.

One of the oldest and most widespread views on this subject is to the effect that drinking-water, especially if it contains much lime, gives occasion to the production of renal calculi. In reply to this theory we may say that, under such circumstances, we merely find an accidental concurrence of two circumstances that are entirely independent of one another, for we cannot see, even *à priori*, how the presence of lime in the water could produce an excess, and, hence, a deposit of uric acid, which is the principal feature in the condition before us.

Pathology.

Pathological Anatomy.

A. *Deposits and Concretions in the Kidneys, the Renal Pelves, and the Ureters.*

Deposits of urinary constituents in the kidneys are met with even in new-born babes, and are called *uric acid infarctions.* They are insignificant in extent, and probably never cause any disturbance in the function of the kidneys. They were described by Rayer, although Schlossberger, in 1842, first recognized their composition, while Virchow, in 1856, most accurately followed out their history in various directions.

The pyramids appear striped, of a yellowish red, brownish, or yellowish color. Their papillæ are full of a thickish, yellowish fluid, which escapes on pressure. The stripes rarely extend through the entire length of the pyramid, and still more rarely do we find the papillæ free and the wider portion only of the pyramid altered as indicated above. In such cases the process of emptying the uriniferous tubules has begun, and the matters discharged from them may also be found in the pelvis of the kidney. These consist of urates—in part urate of soda, but principally urate of ammonia. On the addition of hydrochloric or concentrated acetic acid, the uric acid separates in its crystalline form. This is the crucial test for determining the nature of these deposits. They are deposited in the uriniferous tubules as brownish or brownish red masses, finely or coarsely granular in character, sometimes quite filling the tubules. In the mildest grades we only find the epithelial cells encrusted with finely granular salts.

The same gross appearance is presented by what are termed *hæmatoidin* and *bilirubin infarctions* of the straight tubules. With the latter, it is true, there is always renal icterus. The use of the microscope and micro-chemical analysis protect us against confounding these various conditions.

Virchow urges, with special emphasis, that infarctions of the urates are only to be found in those new-born children whose lungs have been expanded; that, consequently, they do not occur in the still-born fœtus. Upon this is based the forensic value of this sign, it being claimed that, in the absence of other signs, or when they have become indistinct on account of putrefaction, the presence of uric acid infarctions argues, with a degree of probability bordering on certainty, in favor of the life of the fœtus outside of the womb, for this infarction remains visible a long time, even after decomposition has set in. Hodann was able to demonstrate it in the putrid kidney at the end of forty-five days.

With reference to the *causes* of these uric acid infarctions in new-born babes, the majority of writers adhere to the view advanced by Virchow, who regards it as the expression of the increased physiological metamorphosis of tissue which takes place after birth, as the result of the setting-up of the processes of respiration, digestion, and heat-production.

This view, as well as the forensic significance of uric acid infarctions, falls to the ground if such infarctions have been observed even in a single case of a still-born child. The one case which was observed by Hoogeweg was disregarded by Virchow, because the lungs were well filled with air. It may therefore be of some importance here to call attention to a preparation in the pathological institute at Breslau, which dates from Waldeyer's time. It is designated as *Uric acid infarction of the papillæ. Still-born child.* Gynecological Clinic, 1866 (Preparation No. 63 of the year 1866), and should certainly incite us to a careful investigation of this question, which is of such great practical importance, on the basis of a large amount of material.

All observers, however, agree that infarctions are most rare in newly-born children that die soon after birth; that they are more frequent where death occurs during the first day, and most frequent where it occurs between the second and the fourteenth days of life. From that time a steady diminution in frequency occurs till the end of the second month, although in some few instances Virchow has observed uric acid infarctions even in the third to the fifth month.

These infarctions are to be found only in the minority of the kidneys of new-born children (about forty-seven per cent.), so that the conditions for their production are inconstant.

In adults, deposits of urates, in a finely granular form, are not infrequently to be found in single tubules of the pyramids. In one case Frerichs found very little columns with levelled extremities, which consisted of urate of soda. In another case, in a kidney that was the seat of Bright's disease, the same observer found large brown crystals of uric acid, both singly and in clusters of the size of a pin-head, situated in the uriniferous tubules of the cortex and the pyramids, these tubules being filled with amorphous fibrinous clots. The cortical substance felt as if it was strewed full of sand. The pelvis contained glutinous coagulated fibrin, which was also very rich in these crystals. Aside from this, uric acid may be found in dilated uriniferous tubules, generally as urate of soda, in the form of beautiful, generally long, rhomboid columns. Urate of ammonia is very seldom found. Pure uric acid is now and then encountered in renal cysts. Deposits of the urates appear in largest quantity in the kidneys of gouty patients; they are here also found in the intermediate substance and, by the union of smaller concretions, may grow to the size of a millet-seed, or even of a pea.

According to Froriep's observations, deposits of *triple phosphates* also occur in the uriniferous tubules, first presenting the appearance of whitish yellow stripes in the pyramids, then uniting into stones of the size of a hemp-seed and even larger, around which the renal substance then breaks down and suppurates.

Furthermore, deposits of *carbonate of lime* often occur in adults, particularly in the straight tubes, usually appearing in the form of balls and nodular masses ranged alongside of one another, which strongly refract the light. These deposits, called lime infarctions, are especially frequent in older persons, and also when extensive absorption of bone-tissue is taking place. E. Wagner[1] calls attention to the frequency of lime infarctions, sometimes more and at other times less extensive, in the renal pyramids even of young persons who die of small-pox.

To the naked eye, lime infarctions cause the pyramids to appear disseminated with thickly-set parallel stripes. Virchow formerly laid great stress on lime infarctions as a cause of renal cysts. But he receded from this view afterward, as the closure of the tubules by the lime salts is seldom complete. In more advanced disease, lime salts are also to be found in the interstices between the uriniferous tubules.

While the uric acid infarctions of new-born children are confined exclusively to the open tubules of the medullary substance, incrustations of lime salts are more liable to be found in the looped tubules of the medullary substance, and especially in the loops contained in the papillæ. At the same time carbonate of lime is also found in the cortical portion of the kidney. In the Pathological Institute at Breslau there are Bright's kidneys, dating from Waldeyer's time (1871. Sect. Prot. 25), in which numerous yellow spots and points are to be found in the narrow cortical portion, which feel like calcareous masses. These are irregularly formed flakes and fragments which dissolve in hydrochloric acid with active development of gas. They seem to lie in the interstitial connective tissue ; at all events, no distinct location can be assigned to them, as, for instance, in the uriniferous tubules.

---

[1] His Archiv. 1872. p. 114.

Renal sand, *renal gravel*, and *renal stones*, however, are of far more importance than those deposits of urinary constituents imbedded in the renal tissue, and which are generally described as uric acid, lime, or triple phosphate infarctions. While having the same chemical composition, the former are distinguished by their larger size. By renal sand we mean fine powdery deposits, which, when somewhat enlarged, often appear in crystalline form. Renal gravel at the most attains the size of a pin-head, the greater part of the grains being smaller. Renal stones are divided by the French into true renal stones and gravel stones, the latter being able to pass through the urinary passages, while this is not true of the renal stones proper. In practice, gravel stones and renal stones are often confounded ; indeed, no fixed limit can be drawn between the two. Renal stones are sometimes found singly, sometimes in considerable numbers. They differ much in size, varying from that of a pin-head to that of a bean, which is seldom exceeded. At the same time there is no lack of cases reported in literature in which these have reached a far larger size, where they have filled the calyces and pelvis of the kidney, and have weighed from 60 to 100 grammes (two to three ounces). Troja even claims to have found renal stones weighing several pounds. Large stones adapt themselves to the shape of the renal pelvis and the calyces, and thus assume various forms, being often branched like coral. The number of branches varies according as they include a larger or smaller number of calyces. Walter found, in the right kidney of a gouty man, sixty years old, a stone weighing 106 grammes (over three ounces), which had so far assumed the form of the kidney that it resembled a petrified kidney. Aside from these large stones, the number of calculi present is often considerable. I once counted one hundred and fifty of them in a renal pelvis, and they sometimes occur in still larger numbers, although this is rare. As a general rule, no concretions are formed within the ureters. Marcet states that in some few instances he has found the mucous membrane of the ureters covered with a chalky concretion. Most of the concretions pass into the ureters from the renal pelvis, but if they become impacted in the ureter they may very greatly enlarge in size by additional deposits of the urinary constituents, especially of uric acid and the phosphates.

The chemical composition of renal calculi is various.

Those consisting of uric acid are the most frequent. The same relations exist here as with cholesterine in gall-stones. Both exist but sparingly in their respective secretions, but contribute prominently to the formation of concretions on account of their slight solubility. Scheele, who discovered uric acid in 1776, believed that all concretions were composed thereof. This is not true, although five-sixths of all renal stones may be uric acid formations. They may occur at any age, but are especially frequent in persons who have passed middle age, and more particularly in gouty subjects. They are often passed in large numbers. Heller states that he has in his possession 90 grammes (about three ounces) of crystalline uric acid concretions, varying in size from the smallest grain of sand to that of a large long bean, which were passed by an old man within the space of a year. They vary very much in size, ranging from renal sand up to concretions that fill the entire pelvis of the kidney. They present all the colors of uric acid sediments excepting the rose color. We therefore meet with brick-dust colored and vermilion stones, though they are generally somewhat mixed with gray, because a very considerable number of coloring matters become dirty on drying. Sometimes they have a reddish brown, more rarely a greenish color, owing to decomposed coloring matter of the blood which is mechanically mingled with them. Their broken surface is crystalline in small concretions; in larger ones it is generally amorphous, of uniform density, and of a strikingly wooden appearance. This latter characteristic may always lead us to conclude in favor of the presence of uric acid salts. These stones consist of concentric layers, and the lighter or darker color of these layers is accounted for by the greater or less degree of concentration of the urine at different times. These layers are either regular in outline or slightly wavy, and in that case the surface is also somewhat wavy. Renal stones consisting of the urates are very hard, and are only surpassed in this by those consisting of the oxalates. Their specific gravity is about 1.5. The most certain method of recognizing concretions of the urates is by means of the murexid test.

The *uric acid* may constitute the sole ingredient of a concretion, or it may only form the centre thereof, while the outside consists of oxalate of lime. Seeligsohn[1] observed two large concretions, discharged by a girl seven years old, whose shell and middle layer consisted of oxalate of lime, while their kernel was formed of uric acid. More rarely the opposite of this may take place. Sometimes we meet with alternate layers of uric acid and oxalate of lime, more rarely layers of urate of ammonia. Concretions formed of the urates, especially the urate of ammonia, are found much more frequently in nurslings than in adults. Heller observed these soft concretions very often in the kidneys and the ureters of the nurslings in the Children's Hospital at Vienna.

Oxalate of lime is discharged in the form of the octohedra so readily recognizable under the microscope, or of little concretions. These concretions, consisting of oxalate of lime, are never met with in such large quantities as those formed of uric acid. Sometimes only one is discharged, or it may be years before a second one appears. In some few cases oxalate of lime has been observed in the substance of the kidneys; such a condition has been reported by Crosse and Meckel. I have seen it, together with an abundance of blood-corpuscles, deposited on the surface of tube-casts. Oxalic acid calculi are of a dark grayish brown color. They owe this color to the hemorrhages which they cause, whereby the coloring matter of the blood is mingled with them. They are darker than uric acid calculi, and are distinguished by the irregularities of their surface. They are improperly said to be of a mulberry form, whereas they are, more properly speaking, thorny or warty. No other calculus is so rough and irritating to the tissues. The broken surface of these stones always presents an amorphous appearance. There is no proof in favor of the assertion that oxalic acid formations are more frequent in children than in adults. Concretions composed entirely of oxalate of lime are rare; those composed of alternate layers of this salt and the urates are far more frequent. In that case the urates may constitute complete concentric layers, or an incomplete peripheral deposit. It has already been stated that the centre of a mulberry calculus may consist of uric acid, and that more rarely a uric acid calculus may have a nucleus of oxalate of lime. Beale has furthermore observed that a microscopic accumulation of crystals of oxalate of lime may be found in the centre of a uric acid nucleus. In some few instances uric acid concretions have been found in one kidney and oxalic acid concretions in the other.

---

[1] Berlin. klin. Wochenschrift. 1872. Nr. 35.

Cystine or *cystic oxide* calculi are formed of the substance first described by Wollaston in 1810 as cystine. Aside from this, cystine may be discharged in the form of gravel. Concretions of this substance are rare. Statements with regard to their frequency vary very much; they constitute between one and three per cent. of all concretions. According to Owen Rees they are more frequent in dogs. In one case they were found associated with cholesterine. They seem to be more common in England than in Germany. Cystine stones are of a dull yellow color, with scarcely any variations in the shade. On being exposed to the air for some time they are said to assume an emerald green. The specimens in my possession, for which I am indebted to the goodness of Mr. Julius Mueller, in charge of the pharmacy of the All Saints Hospital at Breslau, do not show this change of color, although they have been exposed to the air for more than a year. These specimens consist of a collection of about one hundred concretions, most of which are of the size of a hempseed, though some are as large as a pea, varying in color from a dull whitish yellow to a light amber yellow, their surface being covered with minute warts. They were all obtained from a man between thirty and forty years of age, who was very much reduced as the result of cystinuria, and lately died of inflammation of the bowels. These concretions consist of pure cystine; this is the rule in such concretions. Sometimes they have a nucleus of uric acid. Cystine calculi are far softer than uric acid stones. On fracture they generally present a radiated appearance.

Mueller's determination of the amount of sulphur contained in these concretions placed it at 25.3 per cent. The formula $C_6H_7NS_2O_4$ calls for 26.45 per cent. of sulphur. On microscopic examination of the urine, the well-developed hexagonal tables into which cystine crystallizes could be seen. Now and then cystine crystallizes in rhomboidal tables. It is readily soluble in caustic potash, and crystallizes in beautiful hexagonal tables. It is also soluble in alkalies, mineral acids, and oxalic acid. It can be most readily precipitated from alkaline solutions by the vegetable acids, and from acid solutions by carbonate of ammonia.

Liebig's test for cystine consists in boiling the suspected urine in an alkaline solution of lead. Mueller declares the following to be a nicer and sharper test: The cystine is to be dissolved by means of a little potash lye; the solution when cold is to be diluted with water and some nitroprusside of potassium is to be added, when a beautiful violet color will appear.

In the case from which my specimens were obtained there was nothing abnormal in the proportion of the chlorides and urea, while in other cases of cystinuria a diminution or even disappearance of urea and uric acid has been observed. In this case, too, no hereditary predisposition to the disease could be demonstrated, as has been done in numerous other instances, only it was stated that "diseases of the liver" were hereditary in the family. I am indebted to Geheim Ober-Medicinalrath Woehler for the report of a very interesting case of inherited cystinuria observed by him, in which a mother and both her daughters suffered from the disease. (Toël, in Bremen, has published this case).

Instances of the cure of cystinuria seem to be very rare.

---

[1] Virchow's Archiv. X. p. 230.

Heller reports one in which a girl eighteen years of age remained well after the passage of a large cystine concretion. The statement that cystinuria occurs particularly in the female sex does not seem to be justified. Cystinuria often lasts for years. The majority of cases occur between the ages of twenty and forty. At the same time no entire immunity is enjoyed by any age, not even that of childhood. Hodann removed a large cystine calculus from a boy six and one-half years old, by lithotomy.

Calculi composed of *xanthine* are exceedingly rare. This substance, discovered by Marcet, is very similar to uric acid. The xanthine calculi thus far observed have been free from the admixture of foreign substances, smooth, in part bright and in part dull, of a light brown or dark brown color. Corresponding to uric acid calculi in their hardness, they give no murexid reaction, are soluble in dilute nitric acid without effervescence, and then assume a golden yellow color which becomes orange on the addition of ammonia. They are insoluble in carbonate of potassa.

Concretions of *fibrin* were also first described by Marcet, who gave them their name. They occur as the result of renal hæmaturia with subsequent secretion of fibrin. They may in part be discharged and in part remain in the ureters or the renal pelvis, where they may then be found together with other kinds of concretions. Heller found a tough, fibrinous concretion, almost as large as a walnut, in a kidney which at the same time contained several stones of oxalate of lime, the largest of which were as large as a pea. These fibrin concretions have a dirty white or yellowish brown color, their consistency is that of wax, tough and elastic. They burn with a continuous yellow flame, giving off the odor of burnt feathers. They are insoluble in ether and alkalies, but are soluble in a potassium solution when heated, and may be thrown down in the form of a white precipitate, on the addition of acetic acid, with the development of sulphuretted hydrogen.

Phosphatic stones are found in the kidneys under the form of concretions, consisting of basic phosphate of lime (bone earth) or of ammonio-magnesian phosphate, or of the two mixed. Heller found no basic phosphate of lime concretions (bone earth) among children, though in general they are not so rare. Brodie speaks of two kidneys in his collection that are entirely filled with stones of this kind. The patient had first discharged a little renal stone consisting of oxalate of lime; one or two years later another one was passed, consisting of phosphate of lime. These concretions are white or yellowish white; they vary much in hardness, which is sometimes very considerable, sometimes slight, and their surface may be polished, or rough and dull. Their fractured surface is earthy and amorphous, never radiated. Small concretions, consisting entirely of ammonio-magnesian phosphate, and attaining perhaps the size of a pea, may be discharged with the urine, or may also be found, after death, in diverticula of the kidneys. . They are always of a strikingly white color, their surface is always rough, never smooth, their fractured surface is granular or of a crystalline, radiated appearance. They are never very hard. Sometimes the ammonio-magnesian phosphate constitutes the germ of a concretion. The situation most frequently occupied by the ammonio-magnesian phosphate is that of the superficial layer of concretions, which consist of uric or oxalic acid, and which have lain for a considerable time in the pelvis of the kidney. Concretions consisting of the ammonio-magnesian phosphate and lime phosphate are extremely rare as a primary deposit within the kidneys. They may occur at all ages, but especially in older people. They are much more frequently met with as a secondary deposit constituting the outside layer of various primary stones. Phosphatic calculi cannot be burned, they do not effervesce on being treated with strong hydrochloric acid, and on being treated with a little potash lye (the cold ammonia test) they either develop a little ammonia or none at all, according as they consist of pure bone earth or of a mixture of this with the triple phosphate.

Concretions of *carbonate of lime* are very rare in man. In the kidneys of neat-cattle a considerable number of such concretions are sometimes found at once, varying in size from that of a poppy-seed to that of a hazelnut. Roberts saw an innumerable quantity of carbonate of lime calculi pass with the urine of a man seventy years of age. These were at first taken for prostatic calculi. These concretions resisted the action of fire, and, when pulverized, effervesced on the addition of hydrochloric acid. This case also presented calculi consisting of carbonate of lime, phosphate of lime, and ammonio-magnesian phosphate.[1]

B. *The Pathological Changes produced in the Kidney, the Pelvis of the Kidney, and the Ureter, by Renal Concretions.*

In nephrolithiasis one kidney generally remains healthy, but calculi are generally developed in both sides. In such cases the stones in the two kidneys are sometimes of different chemical composition. Gaultier de Claubry observed uric acid in one kidney and phosphatic concretions in the other. If one kidney remains healthy, it assumes the functions of its diseased fellow, and becomes hypertrophied, in which case it may reach double its normal size. For a long time it was a matter of dispute whether one kidney was more liable to nephrolithiasis than the other, and if so, which one. Morgagni was one of the first who agitated this question, and decided in favor of the greater liability of the left kidney to disease. But the material at our disposal is even now inadequate to the settlement of this question. At all events the right kidney enjoys no immunity from calculous disease. The alterations caused by renal stones differ according to the number, size, situation, and composition of the concretions. But there is certainly also another series of circumstances that come into play in individual instances, whose influence is by no means clear. When deposits in the parenchyma of the kidney take place—as is especially the case with regard to uric acid concretions in the renal substance of gouty patients—these deposits directly irritate the kidney tissue, and give rise to diffuse forms of inflammation. This so-called gouty kidney—which, if not first described by Garrod, was at least most accurately and fully described by him—at first shows uric acid deposits in the pyramids, and later also in the cortex, which finally becomes shrunken and granular. In other cases of nephrolithiasis the features of pyelitis are first developed, combined with more or less extensive participation of the renal tissue, so that in a certain number of instances, as we shall see, the most extreme pyelo-nephritis follows. In arthritis the affection of the pelvis of the kidney is the first in order, if the deposits of urates formed in the kidney are floated into the pelvis and there enlarge and excite inflammation. Concretions of other chemical composition act in the same way as those consisting of uric acid. The anatomico-pathological changes show themselves in very various ways, according to the duration of the affection and the other causes above mentioned which exert an influence thereon. In the lighter grades the only disturbance consists of a more or less highly developed pyelitis. The entire mucous membrane, which is otherwise pale, is found injected, this condition often extending to the renal pelvis. Sometimes this injection is very great, the membrane assuming a deep red appearance, which, in more advanced cases, is accompanied by numberless little ecchymoses. Sometimes the mucous membrane is covered with a fibrinous deposit. The cavity of the renal pelvis and calyces is found filled with a whitish or turbid yellow, more or less thick, mucous, muco-purulent, or purulent mass, the amount of which varies. In this purulent or puriform mass, which is intermingled with more or less urine, stones, or the *débris* of stones, may be found in variable quantities and of different compositions. The greater part of these concretions, unless they are too large, pass through the ureter into the bladder during life. The walls of the ureters take part in the inflammation; they become thickened, and after several large calculi have passed through them they are widened. The escape of renal stones through the ureters seems to be the most common termination of nephrolithiasis. If the stones are large enough, this takes place with all the symptoms of what is called renal colic, which we shall consider later. Sometimes, however, a stone within the pelvis becomes so large that it cannot pass the ureter. It then continues to grow, at first filling the renal pelvis partly, afterwards entirely, and sometimes the calyces too. The urine secreted above the stone is dammed up in proportion to the amount of hinderance offered to its escape. This exercises considerable pressure on the renal tissue, even though a part of the secretion is reabsorbed. The consequences of this pressure may be noticed in course of time, even though it be but slowly and gradually. When there is chronic damming up of the urine, an ever-increasing atrophy of the renal parenchyma from pressure is developed, so that finally nothing of the entire kidney is left but a membranous sac, enclosing a number of cavities that communicate with one another, and represent the former highly dilated calyces of the kidney. The most extreme cases of hydronephrosis may be brought about in this way. If, after the development of the hydronephrotic sac, the obstruction is removed by the subsequent escape of all or a part of the concretions, the sac may shrivel, and its volume be reduced to a very small size. This represents a sort of spontaneous cure of nephrolithiasis and hydronephrosis. If the calculi have escaped, we shall find no evidence on the dead body of how the condition found in the kidney has thus developed itself. Concretions which are impacted in the ureters may there cause inflammation, which may proceed even to rupture of the ureters. If they remain there for some time, they furthermore give rise to an obstruction of the urine, with subsequent hydronephrosis, or to inflammation of the renal pelvis and the kidney itself. Not infrequently purulent pyelitis passes over to the tissue of the kidney itself; ever-increasing portions of the same then gradually break down under purulent degeneration, and finally nothing is left of the kidney but a sac filled with pus and stones, which often attains a larger size than the normal kidney. This sac frequently shows numerous irregular folds. It is formed of the capsule, thickened like a rind, and sometimes of remnants of renal parenchyma which have grown inseparably fast to the capsule, and in which the normal kidney structure has been lost. In such cases the kidney is liable to be extensively adherent to neighboring organs. As the inflammatory process extends to the perinephritic tissue, it often results in extensive suppurations therein, in perforation of the purulent sac itself, and the evacuation of its contents—the pus and concretions—in various directions, especially externally in the region of the loins or into the colon. In some few cases a calculous kidney, which is the seat of chronic suppuration, may be followed by amyloid degeneration of the other kidney and the other abdominal viscera.

---

[1] *Richards*, Dissert. inaugural. 1868. Greifswald.

Symptomatology.

Those patients who are only subject to *renal sand* do not generally make any complaint of suffering. For this reason the symptom may, in some cases, escape the observation of the physician for some time, inasmuch as renal sand is not mingled with the urine every day. Not long ago I treated an old gentleman whose urine I had occasion to examine repeatedly, and in whose case I never found any sediment in the urine. After a while he one day complained of severe pain in the urethra when passing water. I found all the forward portion of it stopped up with urates in the form of sand. After that the urine was always examined immediately after being passed, by the observing patient himself. Sometimes he found a small amount, sometimes an abundance of sand, and occasionally, for some time, none at all. Here the sediments were such as appeared immediately after the passage of the urine, and not only after it had cooled. Some sharp concretions irritated the mucous membrane and caused the pain. Under such circumstances, slight urethral hemorrhages even may arise. Much more frequently, however, these sediments, which generally consist of uric acid, exist for years, not only without pain, but without any perceptible injury to health. In some instances these deposits only appear after certain definite causes have been in operation; in others they are to be found regularly. In some cases of renal stone there is never any passage of renal sand.

But aside from the disturbances in the secretion of urine, as mentioned above, patients who have passed renal sand, in the great majority of instances, do not remember any other symptoms of discomfort accompanying the condition. Now and then they speak of a slight feeling of discomfort, or vague pains in the region of the loins. Notwithstanding all this, however, the evacuation of this powdery form of renal sand is a matter that must not be underestimated, for it is the starting-point, the first beginning of the larger concretions.

The symptoms produced by the larger concretions, by gravel and stones in the kidneys, are generally far more distinct. In many instances they are exceedingly characteristic, but they are by no means always uniformly developed. It should be remembered, first of all, that in some cases of nephrolithiasis all symptoms are lacking. The literature of the subject presents a series of cases in which quite large stones have been found in one or both kidneys, on post-mortem examination, whose presence was never even suspected during life. In other cases symptoms of disease did, indeed, exist, but not such as pointed to an affection of the kidneys. Not infrequently all the manifestations seemed to indicate disease of the bladder, which was nevertheless found healthy, after death, while calculi and the morbid changes produced by them were found in the kidneys. Sometimes but a few vague symptoms are present, which occur in many other diseases, and may be quite accidental. To this class belong dull pains in the region of the loins. Occasionally persons with renal stones bend forward in walking, because it is uncomfortable and painful to them to straighten the spine. In the majority of instances the group of symptoms in nephrolithiasis assumes a more definite form. Let us first consider the *pain*, which often constitutes a very prominent symptom, although not present at all times or in the same intensity. Sometimes the patient is burdened with a continual sense of tension or pressure in the region of the loins, which now and then becomes piercing or cutting, and rises to the most severe paroxysms of pain. In some instances the attacks of pain recur only after months, or even years, and then do not reach a high grade, and have no injurious influence on the general health.

The statement that the pain is dull while the concretions remain within the kidneys, and assumes a pricking or cutting character when they pass into the ureters, does not seem to be sufficiently borne out by observation. But this much is certain, that the pains localized in the region of the kidneys abate or cease in proportion as an intense pain is developed in the direction of the ureters. These severe paroxysms of pain are known and dreaded by patients with renal calculous disease under the name of "renal colic." Hippocrates himself gave a graphic description of these attacks. They develop either slowly or suddenly into one of the most painful affections that exist.

Sometimes patients awake in the middle of the night, from quiet sleep, racked by the most torturing pain, that has seized them as quick as lightning. In other cases the attacks are called forth by active or passive movements of the body, or exertions, such as riding, driving, jumping, running, or by labor of the most various kinds, even by such acts as sneezing, coughing, etc. During the attacks people bend up double, or endeavor to find relief from the pain by lying on the painful side with their thighs drawn up. Every movement is torture to the patient, as it increases his pain. The pain is not confined to the region of the loins and the course of the ureters; it spreads over the entire abdomen, or radiates to the breast, penetrating to the shoulder-blades, or runs along the false ribs or the crest of the ilium. It is often accompanied with pain in the testicle of the affected side, or this may radiate to the extremity of the glans penis. If the attack lasts long the testicle is not only sensitive, but it swells. Prout observed several cases in which pain with swelling of the testicles was one of the most severe symptoms of renal colic. Numbness—a feeling as if the limb of the corresponding side had gone to sleep—is frequently present. During the attacks exacerbations and remissions alternate. During the latter the patient, exhausted by suffering, obtains a certain amount of rest, which is unfortunately soon disturbed by a new paroxysm of pain more severe than ever. The suffering often reaches an extreme grade. It is so overwhelming that it may paralyze the most powerful man. He becomes as helpless as a child, and trembles as if in mortal terror, while heavy sweat-drops stand upon his forehead. The pain extends to the thigh of the affected side; the testicle becomes painful, and is drawn up against the abdominal ring. Fainting-fits and general convulsions have been observed as the result of renal colic. These pains are often accompanied by *gastric manifestations* of various degrees of intensity, from the mildest grades of nausea to repeated violent vomiting of watery bilious matter. The gastric manifestations sometimes play a very prominent part, so that the entire attack has a specially gastralgic character. Elevations of temperature of a tolerably high grade are often developed. The pulse grows small and greatly accelerated; the frequency of respiration is also materially increased. Pregnant women often abort under these circumstances. In Troja's work we find the report of an observation in which a woman with calculous disease aborted fourteen times, always during the eighth or ninth month. A patient of Simon's aborted twice during attacks of renal colic, once at the end of the fourth month and the other time at the end of the fourth week of pregnancy.

Great significance is to be attached to the *composition of the urine and the behavior of the urinary secretion during the attacks.* The pain extends to the bladder and a painful spasm of the bladder often arises, with severe strangury or ischuria. The urine, which is often only voided drop by drop at such a time and is always scanty, is red, brown, or blackish, exceedingly loaded with blood, often mingled with mucus or pus. It causes severe burning in the urethra. Sometimes an entirely colorless urine, as clear as water, has been observed during the most intense paroxysms. This occurs when only one kidney is affected with calculus, and its ureter is obstructed, while the other kidney is healthy, performs its functions normally, and its ureter is unobstructed. If both kidneys have calculi, or if the individual has but one kidney and this is so affected, then there may be complete cessation of the escape of urine, accompanied by all the conditions belonging thereto, provided that both ureters are occluded, or the single ureter where but one exists, and the escape of urine is entirely prevented.

If the obstruction to the escape of urine is not removed in time, death will follow, with coma and convulsions, generally, at the latest, ten days after the beginning of the attack. A considerable number of such cases exist in literature. Thus, Brodie reports a case observed by Travers, in which both ureters were entirely closed by stones at their point of origin at the renal pelves. In some instances, however, recovery seems capable of occurring even after long continued anuria.

In a widow, sixty-three years of age, who had suffered for fifteen years with symptoms of stone, Salgado[1] witnessed recovery following the escape of a calculus the size of a bean, with much gravel and an abundance of urine, *after complete anuria had lasted for thirteen days.*

---

[1] Extract in Schmidt's Jahrb. 158. p. 139.

I find a very remarkable case reported by Owen Rees:

On making a post-mortem examination of a man whose blood was more impregnated with urea than that of any case of Bright's disease which he had examined in that particular, it turned out that there was but a single kidney, whose ureter was obstructed by a stone. The patient had been in the possession of his senses up to the last moment. Unfortunately, nothing is stated with regard to the evacuation of urine. The only explanation of the case we can give is that the position of the stone was such as to admit of the urine flowing down alongside of it.

In some instances it is remarked that the anuria ceases after a time, to return again later.

Dittell[2] communicates a very interesting instance of this kind. A man, thirty-nine years of age, was suddenly attacked with severe colic. The urine was turbid and foul smelling. On the fourth day the urine was bloody; the same was true a month later. No renal gravel at any time. Severe pains in the lumbar regions, radiating towards the scrotum. A *diminution in the quantity of the urine*, which in a few days increased to *complete anuria*. Sopor alternating with conditions of great exaltation, frequent vomiting, no fever. These symptoms, no definite cause for which could be found, disappeared after a few days, to return again at the end of a a month and terminate fatally, with convulsions. On post-mortem examination a horse-shoe kidney was found. One of the two renal pelves was found entirely filled with a large renal stone whose prolongations extended into the calyces; the other pelvis was capable of being shut off from the ureter by the descent of a concretion which filled and closed one of the three calyces.

Some few cases exist in literature in which *complete anuria* occurred with a fatal termination, when only one kidney was found to be capable of performing its function on account of nephrolithiasis, while the other was found quite healthy.

The attacks of severe renal colic described above, and which occur when the communication between the renal pelvis and the bladder is interfered with in such a manner that the escape of urine is entirely or almost entirely prevented, are due, not so much to the irritation of the mucous membrane by the concretion, with which they stand in no direct relation, but rather to the distention which the urinary passages experience by the urine accumulated behind the concretion. This fact was made prominent by Prout and again recently by Traube. The colic is

---

[1] Diseases of the Kidney with Albuminous Urine. German translation. 1852. p. 52.

[2] Anz. der k. k. Gesellsch. der Aerzte in Wien. 1872. No. 2.

called forth by the futile peristaltic movements which take place from time to time in the abnormally distended urinary canals. These paroxysms last until the obstruction is in some way relieved. During their passage through the ureters the calculi, becoming wedged in, may also cause ulceration of the walls of the ureters with perforation of the same, whereupon, as a general rule, death very quickly follows with symptoms of peritonitis.

Allan Webb[1] narrates such a case. It occurred in the person of a robust European sailor, who died with symptoms of peritonitis, after having been sick for eight days with pain in the right hypochondrium. The ureter and vermiform appendix were found ulcerated and adherent to one another and to the surrounding structures. A large amount of pus had escaped from the ulcerated ureter into the abdominal cavity. Both kidneys were very much diseased; both contained numerous abscesses; the left kidney contained four stones, and the bladder also contained a stone.

But such cases may also run a chronic course.

J. P. Frank tells the story of a nun in Cremona, in whom the left ureter, which was grown fast to the wall of the abdomen, was eaten through by a stone in such manner that after the latter had given rise to an abscess it worked its way outward through the abdominal muscles.

Such occurrences, however, are but rarely met with. In the majority of instances, concretions that have once entered the ureter pass through it. When the obstacles to their passage are successfully overcome, the attack of colic is at an end. In one set of cases this occurs at the end of a few hours, in another set these attacks last for days and even weeks. Then the progress of the imprisoned stone through the ureter can sometimes be followed by the changing seat of the most intense pain and by certain modifications in its character. It is well known that the ureter is narrowest at its lowest portion, where it opens obliquely into the bladder. This is the point at which concretions are usually retained the longest, and here, too, by causing the most complete retention of urine, they produce the worst symptoms. But then, too, all troubles often suddenly cease, as at one blow, when, after the last exacerbation, the concretion overcomes all obstacles and drops into the bladder. The greater the obstacle to the escape of urine presented by the stone, the more intense will be the attacks of colic. If the urine can pass down into the bladder alongside of the calculus, then the backset and the colic do not attain a high grade. In a certain series of cases the attacks of colic do not end suddenly, but slowly and gradually. There are cases in which stones of but slight consistency crumble into smaller particles of gravel, or into sand within the ureter. The longer nephrolithiasis lasts, and the more frequently the attacks recur, the milder the renal colic often grows, though this is, unfortunately, by no means always true. This depends on the fact that the accumulation of fluid accompanying each attack results in enlarging the calibre of the ureter. If the concretions now entering the ureter are larger than those previously passed, then, it is true, the dilatation is of no significance. In some persons attacks of renal colic appear periodically and recur at regular intervals of time, this being sometimes very strikingly true.

---

[1] Pathol. indica. Calcutta. 1848. p. 220.

Sometimes renal stones escape from the kidney through some traumatic accident, without being accompanied by an attack of renal colic. Brodie alludes to a very instructive case of this kind.

A patient who had several times observed a bloody discoloration of his urine received a severe blow on the occasion of the upsetting of his wagon. He afterward had the most urgent desire to pass water, but could not accomplish it. After severe straining the obstacle gave way, and a stone, apparently of the shape of the renal pelvis, was expelled with great force into the vessel.

As a matter of course, these attacks of renal colic are only observed when concretions attempt to pass the ureters which are too large for them. If the concretions are small enough to pass the ureters without obstruction, as we have seen above in case of renal sand or the smaller forms of gravel, or if they are so large that they cannot leave the renal pelvis, then, of course, renal colic, as here described, cannot occur. In view of the uncertainty so often attending the other symptoms, we are then obliged to fall back on careful and repeated examinations of the urine.

The composition of the urine varies materially, according as pyelitis or nephropyelitis has been developed, in consequence of the presence of the renal stones, or as these organs are not yet the seat of consecutive anatomical changes. In the latter instance all that we can expect to demonstrate is the presence of single crystals or particles of the urinary components constituting the renal stones. One would find himself very much mistaken if he supposed that he must always find a sediment of the urinary constituents forming renal stones in the urine of persons thus affected. It is often only after repeated examinations, in which time should always be allowed for any sediment the urine may contain to settle, that we can find the ingredients of the calculi, especially in concretions consisting of uric acid, oxalate of lime and cystine. These sometimes appear in little crystals, and indeed in such small quantities that they can only be discovered by the aid of the microscope (oxalate of lime, cystine); at other times they form quite an abundant sediment, especially if consisting of uric acid or urate of ammonia. Especial importance is to be attached to those sediments which appear immediately after the evacuation of the urine, which, therefore, exist as such in the urine as opposed to those which are thrown down only after the urine cools. Aside from these powdery deposits consisting of urates, larger grains of gravel are often seen varying from the size of a poppy-seed to that of a pin-head. They often escape observation in the midst of these powdery sediments. They can be got hold of most easily by shaking up the sediment found at the bottom of the vessel. Then the larger grains, being the heavier, fall to the bottom again first. Oxalate of lime, cystine, the phosphates, especially phosphate of lime and the ammonio-magnesian phosphate, are also sometimes voided with the urine in the form of such little gravelly kernels. With the escape of such concretions a final cure is sometimes effected, no new ones being afterwards formed. The permanent disappearance of all the deposits hitherto observed in the urine admits of this conclusion. In patients with renal calculus, even at times when the urine contains no sandy or gravelly sediment, it is not rare to find a very abundant deposit, consisting principally of the epithelium of the renal pelves and calyces.

In a female patient whom I have had under observation for several years, and who recently, after a severe attack of renal colic, voided a number of larger concretions, I found no other symptoms during the earlier period of her illness but occasional severe attacks of gastralgia, and I at the same time noticed an abundant sediment in her urine. This consisted of the characteristic epithelial cells of the renal pelvis, which were encrusted in varying degrees with uric acid. In some of them the nucleus was still visible, in others it was so strongly encrusted that it could not be seen. The first class had a light yellow or reddish yellow color, the latter class a reddish brown or brownish black color. The numerous epithelial cells lying in the field of the glass presented a beautiful picture, resembling in their tones of color the scales from the butterfly wings of the Papilio Janira. The fact that this incrustation was not accomplished after the evacuation of the urine was proved by causing the patient to pass water into a warm glass, immediately filtering it, and examining the sediment on the filter. In order to see whether there was any renal sand or gravel present, the sediment was suspended in distilled water, but no falling of heavier kernels of gravel took place. It was not until after repeated examinations that I succeeded in finding single little clusters of crystals consisting of uric acid and gravel granules of the same. There was scarcely any pavement epithelium, and the few cells present were not encrusted. The urine was highly acid, contained a very small number of pus (lymph) corpuscles, and an exceedingly scanty allowance of red blood-globules.

The greater the irritation caused in the kidney and renal pelvis by concretions, which is especially great in case of the rough, thorny, and very hard oxalate of lime stones, the higher will be the grades of pyelitis and pyelonephritis developed, and the more abundant will be the purulent admixture in the urine, together with which renal sand or gravel or small fragments of stone may be found. In the same way a bloody admixture with the urine may also be found, provided that the communication between bladder and kidney, through the ureter, is open. If, after extensive suppuration of the renal parenchyma, the entire kidney with its pelvis becomes transformed into a sac filled with pus, crumbs of calculi and concretions, then, in a certain number of cases, another symptom is developed, viz., a *tumor* perceptible to objective examination. The tumor can be perceived by the touch, can be defined by percussion, and can sometimes be recognized on inspection by the increased width and greater prominence of the lumbar region on the side affected. The renal tumor can be felt as a smooth or slightly nodulated mass, generally painful to the touch, and distinctly or indistinctly fluctuating. On percussion it gives a dull flat, or dull tympanitic sound. Now and then a growth in the size of the mass may be observed, with increase in the tension and painfulness of the tumor, and the group of symptoms characteristic of renal colic. This occurs when a concretion has become so wedged in as to interfere with the escape of the contents of the sac. If the other kidney is healthy, then entirely normal urine may be passed, instead of the abnormal fluid found when the communication between the sac of pus and the bladder is open; a circumstance which, as we shall see, is of great consequence in establishing the diagnosis of one-sided renal affection. If the obstruction to the concretion ceases, and the contents of the purulent sac can again escape, then the attack of colic abates, the tumor diminishes in size, and the urine once more grows turbid, containing pus or blood and mingled with gravel and concretions. The irritation being continued, the suppurative process sometimes extends beyond the kidney, whereby the symptoms are materially complicated. Inflammation is developed in the perinephritic connective tissue, and the pus sometimes descends along the internal iliac and psoas muscles to the inguinal region. The perinephritic process may stop here, and after the pus is discharged it may heal.

I observed such a case in one of my patients. He was a man thirty-four years of age, whom I had treated in 1864, in the All Saints Hospital at Breslau, for calculous pyelonephritis of the right side, with a distinct tumor and copious pyuria, although at that time without any well-marked renal colic or the passage of larger concretions. He returned to the institution in 1867 with severe pains in the right lumbar region, which extended downward along the ureter. Finally, an abscess as large as a child's fist was developed in the right inguinal region, on the inner side of the thigh, after the evacuation of which the pain at once diminished and soon ceased. The wound healed and he left the hospital materially improved. He did not return till 1872, and died in Professor Lebert's[1] medical clinic in 1873, in consequence of perforation into the colon of the purulent sac formed by the right kidney. I shall refer to this interesting case again hereafter.

In other analogous cases, however, extensive ichorization follows as the result of such perinephritic suppuration, which, as a rule, speedily leads to death, generally by septicæmia, and sometimes almost suddenly with symptoms of collapse. In other cases of nephrolithiasis, after adhesion has taken place between the kidney and perinephritic tissue on the one hand, and the muscles of the lumbar region on the other, *perforation takes place with the escape of the pus to the outside.*

---

[1] Compare Inaugural Dissertation of *O. Rosenbach*, 1873.

I had occasion, in 1868, to observe a patient with this kind of lumbar renal fistula for several weeks. I then obtained the following data from the patient, a very intelligent merchant. He was a temperate man, of medium height, not corpulent, and was taken sick suddenly in June, 1862, being then thirty-four years of age. He could assign no cause for the attack. No instances of calculous disease are said to exist in the family. While out walking, he was suddenly seized with such distress in the abdomen that a cold sweat broke out on his forehead. After some hours his symptoms were ameliorated, but the pains still continued the next day. During the autumn of the same year he had another similar attack. After lasting for several hours, it ended in vomiting. Since that time they have recurred every eight or ten days. In the spring of 1863 they ceased until autumn, and returned as before. In July of 1864 the patient experienced one of the most severe attacks he ever had, at which time there was considerable swelling of the abdomen. At the same time pains in the region of the left kidney set in, and suddenly abundant quantities of pus appeared in the urine, the act of micturition being very painful, while the other manifestations disappeared. His subjective condition improved after that, though there still remained a tendency to swelling of the abdomen and flatulence, and sometimes there was pain in the left inguinal region, which was especially liable to recur under the influence of taking a slight cold. Otherwise the patient was pretty well for three years; after that, a stoppage of urine suddenly occurred. This trouble also vanished after the expulsion of two concretions, one of the shape of a heart, and the other of a bean, of a grayish marbled appearance. From this time on renal gravel was passed in abundance. At the same time distressing attacks of "gastric cramp" were developed. Still later a great number of pains and abdominal troubles of various kinds set in. Early in 1868, a swelling in the region of the left kidney appeared. It is said that this several times spontaneously diminished in size. Finally, the tumor opened itself, with the discharge of a large amount of pus. A short time afterward I observed the patient for several weeks. I have not seen him since then, and have only learned that he died in 1873, that is, after the disease had lasted for eleven years.

Post-mortem diagnosis: chronic cheesy broncho-pneumonia, with the formation of cavities; ulcers of the larynx and the intestines; *calculous nephritis of the left side; perinephritis;* diphtheritis of the bladder and the ureters; cloudiness of the liver. When I saw the patient, in 1868, he had as yet no phthisis. This was only developed as a terminal process.

In some cases the concretions themselves are discharged through these fistulas. Occasionally the opening of an abscess in the lumbar region is followed by recovery, the wound closing after a longer or shorter time. In a number of instances calculous pyelonephritis has been followed by perforation into the colon, a pathological occurrence which seems to have been known to the ancients.

In recent times Bright[1] has given an account of a man, about forty years old, who passed pus by the urethra and the rectum. A stone lay in the left renal pelvis, which had been growing for about twenty years. The communication between the abscess and the colon was of about the size of a goose-quill; the ulceration had extended to the lumbar muscles. Bright states that one or two specimens of this sort may be seen in the Museum of Guy's Hospital. J. W. Ogle[2] observed a woman, thirty-one years of age, who was taken sick with fever, pain in the abdomen, and stoppage of urine. There was a round, hard tumor in the lumbar region, besides pus in the urine, fever, and night-sweats. In spite of this a temporary improvement took place. The patient left the hospital. Six weeks after, diarrhœa set in, and the patient also voided a stone consisting of uric acid and oxalate of lime. After feeling well for several months, she was again seized with fever, pain in the bowels, vomiting, and diarrhœa, and died at the end of three weeks. At the autopsy, extensive adhesions were found between the colon, the stomach, and the liver, and especially between the sigmoid flexure and the upper part of the enlarged right kidney, in which a cavity was found containing three stones as large as a pea, and communicating with the colon. The left kidney was normal.

In the case which I have already mentioned (p. 717), and which died in Lebert's clinic, there was a fistulous opening 13 ctm. (four inches) above the ileo-cæcal valve, which communicated with the right renal pelvis and was nearly a centimetre in size (one-quarter of an inch). The right kidney was separated by a sac containing a large number of cavities filled with a grayish, unctuous mass. The patient suffered with profuse diarrhœa, which has also been found true of most analogous cases. Streaks of pus were found in the stools, and as there was no demonstrable intestinal disease, it was altogether probable that this originated in the kidney. No urinary ingredients could be found to exist in the dejections. This was natural enough, as all the secreting portions of the diseased kidney were destroyed. The urine smelled intensely of sulphuretted hydrogen, a silver catheter was blackened by the urine. The solid constituents of fæcal matter were not found in the urine, and the same is true of other similar cases. The contents of the kidney could therefore pass into the intestine, but the reverse of this was not true. For this reason, too, the urine could not be colored by coloring matters introduced into the bowels. The relation of things here is probably the same as that which effects the closure of the ureters by their oblique passage through the walls of the bladder. The urine, in addition to some well-preserved pus-corpuscles, contained masses of detritus and bacteria. After some time a strong ammoniacal smell was developed in addition to the sulphuretted hydrogen smell, and during the latter days of life the ammoniacal smell alone was perceptible.

---

[1] Guy's Hosp. Rep. Vol. IV. 1839. Case 8.

[2] St. George's Hosp. Rep. Vol. II. p. 346.

All these cases have the following characteristic symptoms: *the escape of pus with the fæcal dejections and simultaneous pyuria, which is demonstrably occasioned by renal disease.* In one case which is reliably confirmed there was a fistulous communication between a calculous kidney and the stomach.[1]

During the life of the patient portions of food, poppy seeds, etc., passed off with the urine. Post-mortem examination showed adhesion between the stomach, the right kidney, and the liver. A probe could be passed from the stomach, through an opening in its posterior wall close to the pylorus, into a large abscess of the upper portion of the right kidney, in which many fragments of calculi as well as grape and apple seeds were found. The renal pelvis contained two large calculi.

In rare instances perforation takes place in two directions, into the colon and through the lumbar region to the outside world. Such a case is described by Peter Frank. Here the urine, wind and fæcal matter passed out both at the anus and at the fistulous opening.

Complications and Sequelæ.

a. *Arthritis.* The old writers (Sydenham, Boerhave, van Swieten, and others) laid great stress upon the contemporaneous occurrence of gout and renal stones, which was regarded by some as constituting the rule. Erasmus wrote to a friend: "I have renal disease, and thou hast the gout; we have married two sisters." The inactivity and the recumbent posture to which gouty patients are doomed by their sufferings were even, formerly, looked upon as among the predisposing causes of nephrolithiasis. We know from more recent investigations with regard to gout, especially those of Garrod, that an accumulation of uric acid takes place in the blood, that is, that there is a uric acid diathesis. Although, as I stated above, no excess of uric acid is necessary for the formation of concretions composed of this substance, yet such an excess naturally favors their formation within the organism. I have already given a description of the gouty kidney under the head of Anatomico-Pathological Changes. In Germany we seldom have an opportunity to make an anatomical study of gout.

---

[1] Oesterr. med. Wochenschr. 1844. No. 5.

In 1872 I had the first opportunity that has ever presented itself to me during all my hospital service of making a post-mortem examination in a case of arthritis. The highest degree of renal changes was found to exist, besides the most extensive gouty deposits in the large and small articulations, in the cartilage of the ear and the tissue of the skin. He always had some open tophi on his fore-arm, which furnished ever ready material for demonstration to the eye and for the application of the murexid test. At the time of his death the patient was over sixty years of age. He had always been in most needy circumstances. As long as he was under my observation he never had any uric acid formations in his urine.

Garrod, who has had such an extensive experience in gout, has shown that gout and gravel often occur in the same individual. Sometimes they exist simultaneously, but more frequently at different periods of life, it appearing that those who in later years are attacked by gout suffered from renal gravel in their youth.

b. *Scrofula and tuberculosis.* Meckel brings the two diseases into the most direct causal relation to one another, stating that the development of calculi in the renal pelves is only to be found in subjects who have no predisposition to typhus, intermittent fever, albuminuria, and the like, but who have been or are now subject to scrofula. Other authors make similar statements. Rachitis, too, has often been thought to have some connection with calculous disease; but there is no strict evidence of this. The figures given by Rilliet and Barthez are far too small to admit of general conclusions being drawn from them, and they are, furthermore, confined to the age of childhood. They observed eight children in hospital with renal gravel. Tuberculosis appeared as a concomitant disease in four cases, and they all had tuberculosis of the encephalon. One child was in the incipiency of acute tuberculosis, and died of gangrene of the lungs. Of the other three children, one died of scarlatina, with cerebral attacks, one of typhus, and one of dysentery.

Pulmonary phthisis is not infrequently developed secondarily, as the result of chronic suppuration in the kidney, as has been observed in calculous pyelonephritis. Long continued, suppurative, calculous pyelonephritis of *one* kidney, just like other chronic suppurative processes, now and then gives rise to

c. *Amyloid degeneration of the other kidney* and of the other abdominal organs.

d. *Calculi in other organs* often complicate nephrolithiasis, this being most frequently the case with stone in the bladder. It is well known that the majority of urinary concretions are developed in the kidneys, and vesical calculi are formed by the enlargement of concretions that have descended from the kidney to the bladder, and which cannot pass through the urethra. P. Frank narrates a case in which, in addition to renal stones, concretions were found in the lungs and liver. Renal stones and gall-stones are not seldom found in the same individual. The presence of gall-stones, together with renal colic, may render the diagnosis very difficult.

e. Finally, nephrolithiasis may be complicated with various other affections. Complication with disease of the stomach is of special importance. I stated above that in renal colic it was not at all rare to find gastric disturbances quite prominent, such as nausea, vomiting, and pain in the epigastrium. If renal stone is complicated with disease of the stomach, then it often requires the most careful weighing of the symptoms in order to escape errors in diagnosis.

This may be illustrated by a case that came under my observation, in which nephrolithiasis was complicated with a perforating ulcer of the stomach, and the latter caused death by opening a large gastric artery. A woman, fifty-three years old (received into the All Saints' Hospital at Breslau, in November, 1865), had suffered since she was thirty years old with attacks of nausea and watery vomiting, which recurred every four or five weeks. After this condition had lasted for ten years, the attacks always passing off quickly, pain in the region of the stomach and the vomiting of food appeared as additional symptoms. She never vomited blood. The attacks came seldom; sometimes an entire year elapsed between them, but then they were of such intensity that she had several times before been obliged to take refuge in a hospital. For the past four years a decided remission in these attacks had occurred. They were reduced to attacks of gastric pain of short duration. Four weeks previously the attacks had grown more frequent and severe, assuming the following form: severe pain in the region of the stomach, followed by vomiting, without headache; afterwards severe pain in the small of the back, in the region of the first three lumbar vertebræ, from which point the pains pass to the sides, "as if they were about to break through her ribs." During the attacks there was complete loss of appetite; at other times, too, the ingestion of warm food caused nausea. The bowels were regular. In the hospital I observed the attacks, which occurred several times a day, and lasted from five to eight hours. They followed one another so frequently that the patient was hardly ever free from pain. The pains in the back and stomach ceased at the same time. While in the hospital she never vomited, but was nauseated during the attacks. Aside from this, the patient had for about eight years observed a tumor in the right side of the abdomen, and for six or seven years a turbid quality of the urine. Within about a year the patient had become much emaciated. The epigastrium was painful on pressure, as far down as the navel, and during the attacks was spontaneously painful. Three fingers' width below the margin of the ribs on the right side, and to the right of the outer border of the rectus abdominis muscle, one could feel a tumor at the lower border of the liver, which seemed as if it were connected with this organ. It gave a dull, tympanitic sound on percussion. The tumor was painful to pressure only on the last day of her life. It seemed as if it could be reduced in size by pressure. Sometimes it felt soft and apparently fluctuating; sometimes it presented a tense, stretched, somewhat nodular surface. The daily quantity of urine varied from 600 to 1,000 c.c. (19 oz. to 32 oz.); during the attacks it sank to from 200 to 300 c.c. (6 oz. to 9 oz.) during twenty-four hours. During this time it consisted almost entirely of pus, while in the intervals of an attack it only showed a very copious purulent deposit. No gravel or concretions were ever observed in this sediment. The urine was always strongly acid. While in the hospital she had no appetite whatever, and was constipated. The subcutaneous injection of morphine alone sometimes brought her relief for a short time. On the afternoon of the 7th of December, 1865, she was suddenly seized with an attack of great distress and oppression, combined with temporary loss of consciousness. I found her fearfully exhausted—completely anæmic. She complained of the most violent pain in the region of the kidneys. The abdomen was soft, and in general not very sensitive. The tumor could be most distinctly separated from the liver, as to its boundaries. There was no vomiting—one involuntary passage from the bowels of black, tarry matter. She had several attacks of syncope, in one of which she died that evening.

On post-mortem examination I found the stomach in a vertical position and distended with black, tarry, clotted blood. On the posterior wall of the stomach, on the lesser curvature, and not far from the pylorus, there was an ulcer measuring four centimetres in height by two in width (an inch and a half by three-quarters of an inch), the edges of which were mostly sharp, but perfectly undermined. The floor of the ulcer was formed of connective tissue and of the pancreas which was grown fast to it. In the left upper corner of the ulcer I found the eroded artery.

The tumor under the liver was firmly adherent to it and to the beginning of the ascending colon. At the same time it was nowhere covered with loops of intestine. The tumor, which was formed of the right kidney, measured 16 ctm. from above downwards, 12 ctm. in its greatest width, and 5 ctm. in its greatest thickness (6¼ by 4¾ by 2 inches). The renal parenchyma was entirely destroyed. The tumor consisted of two sacs, a larger one above and a smaller one below, incompletely separated by wall-like septa and bands. The contents of this cavity consisted of a grayish white, thick, purulent mass. The wall of the sac measured some millimetres in thickness. On its inner surface there were some very little insignificant portions that reminded one of renal tissue. The pelvis of the kidney, 4 mm. thick ($\frac{3}{20}$ of an inch), was filled with a stone of chestnut-brown color, consisting of uric acid, which was adherent to the posterior wall of the renal pelvis. The remainder of the mucous membrane of the pelvis, as well as of the ureter, was pale, the right ureter wide, its opening into the bladder free. The left kidney was a little enlarged, and showed a moderate number of little superficial cysts with glutinous contents. Its parenchyma was pale and relaxed. The bladder was healthy.

Diagnosis.

The diagnosis in a case of nephrolithiasis, aside from determining whether renal calculi exist at all, should settle the question of the variety of concretion present, and whether one or both kidneys are involved. When considering the therapeutics of this affection, we shall see that the accurate solution of these questions is of the greatest practical consequence. Two elements come into play in the construction of a diagnosis: one is the examination of the urine, and the other consists of the symptoms caused by the presence of concretions in the apparatus charged with the preparation and expulsion of the urine. The examination of the urine is all the more important, because it often gives us the only evidences in favor of nephrolithiasis at a time when therapeutic interference proves of the most avail. In treating of the symptomatology, I called attention to the fact that, under these circumstances, the urine often contains gravel consisting of the ingredients of the urine which compose the concretions. The diagnosis is rendered certain if actual little concretions appear in the urine. The easiest method of finding these has already been indicated on pp. 715 and 716. If the condition is complicated with calculous pyelitis, mucus, pus, blood, and sometimes the characteristic cells from the pelvis of the kidney, will be found mingled with the urine in variable quantity. The fact that larger concretions have been developed in the kidney is proved if these escape with the urine, either entire or in fragments, accompanied by the symptoms of renal colic. At the same time it must, of course, always be proved, especially if the renal colic is on the right side, that we are actually dealing with a case of renal stone. For instances do exist in literature, although they are extremely rare, in which, a communication existing between the gall-bladder and ureter, biliary calculi have found their way into the urinary bladder. In one of these cases nine gall-stones, and in another two hundred, were passed with the urine during a week's time. Generally, indeed, the presence of highly icteric urine without icterus of the body, would be sufficient to lead to such a diagnosis. But if we have dropsy of the gall-bladder opening into the ureter, this diagnostic sign will fail, and the matter can only be determined by ascertaining the condition of the kidney. The mere presence of cholesterine in the urine is not conclusive evidence. Murchison[1] observed a large quantity of cholesterine and pus in the urine of a man who afterwards died of calculous pyelitis, and in whom there was no communication between the urinary and biliary passages. But sometimes renal stones are developed to such a size that they cannot leave the pelvis of the kidney, to say nothing of passing the ureter, and where, during the entire course of the illness, attention is never called to the existence of nephrolithiasis by the presence of renal sand or gravel—where, in fact, the disease is never suspected during life. If the entire group of symptoms is not clearly enough defined, we must be careful not to lay undue stress upon single signs. Thus, in the diagnosis of nephrolithiasis, great importance has been attached, and that not without reason, to periodically recurring renal hemorrhages. For the most frequent cause of renal hemorrhages is the change of position of a calculus developed within the kidney, and the wounding of the tissues caused thereby. But we must remember that, on the one hand, certain cases of renal calculus run their course without any hæmaturia, and, furthermore, that the hæmaturia itself may introduce a source of error, since the passage through the ureter of fibrinous clots caused by the hemorrhage may give rise to attacks which are completely analogous to those of calculous nephrolithiasis (indeed, as we have seen, there are actual fibrinous concretions). In this manner other affections combined with hæmaturia may be confounded with nephrolithiasis. Any renal hemorrhage that takes place slowly may give rise to the formation of clots in the infundibula. This may take place in the hemorrhages following renal cancer. I spoke at greater length on this subject when describing renal cancer (pp. 674 and 676). I here make room for but one instance from the rich experience of Todd.

---

[1] Path. Transact. Vol. XIX.

In a man a little over sixty years of age, a tumor of the right kidney could be distinctly felt. It was first discovered when the examination of the abdomen was rendered necessary by the occurrence of a painless hæmaturia. On the passage of the clots through the ureter the signs of renal colic appeared. At one time it made the impression of there being stones wedged in at the end of the ureter. In spite of the discharge of the clots and the healing of the vesical catarrh which they occasioned, the tumor grew more and more. Several such attacks appeared. The urine showed nothing abnormal. Death followed in a few months. Post-mortem examination revealed an enormous cancer of the right kidney, with cancer of both pleura and the mediastinum.

Aside from renal cancer, other diseased conditions of the kidneys, the renal pelvis, and the ureters may give rise to symptoms quite analogous to those of calculous renal colic. Thus, the diagnosis may be rendered exceedingly difficult, and in certain stages of the disease impossible. Among these conditions are gouty and caseous inflammations, parasites in the renal pelvis, especially echinococci, etc. The diagnosis is peculiarly difficult if we have a combination of any of these forms of disease with nephrolithiasis, which is by no means a rare occurrence.

It is furthermore important to distinguish between attacks of gastralgia, renal colic, and the colic resulting from gall-stones, the more so as renal stones are by no means always accompanied by characteristic renal colic, but sometimes merely occasion attacks of gastralgia, or produce a combination of various conditions of this kind.

Especial care is necessary in judging of those cases of nephrolithiasis in which the patients complain only of bladder symptoms. But if the urine is bloody and purulent, with pain in the loins, especially if this is localized in one side, the question of whether there is not a stone in the kidney must arise, even though the individual may refer all his symptoms to the bladder.

The order of consecution in which the symptoms arise determine the question. In an original affection of the bladder the symptoms are all referable to this organ long before pain in the loins appears.

The diagnosis of whether but one kidney is affected, and, if so, which one, is not only of prognostic significance, but has of late assumed new prominence since the action of Simon (see Therapeutics) has raised a discussion as to the propriety of extirpating the diseased kidney for the cure of nephrolithiasis. The anatomical experience that calculous disease generally attacks but one kidney, and the fact that the other kidney vicariously performs the function of the diseased organ, argue in favor of the extirpation of the calculous kidney, which is functionally of very little or no avail, and only injures the organism. The fact of the colic pains occurring only on one side justifies the conclusion that the kidney of that side is diseased, but not that the other kidney is entirely healthy, for the latter may yet be the seat of a series of little concretions which at the time give rise to no objective signs. At the same time there is *one symptom which argues with the greatest probability in favor of the healthy condition of one kidney, viz., when, during the attack of renal colic—in which the ureter of the diseased kidney is so obstructed that none of its secretion can enter the bladder—perfectly normal urine is passed, instead of the abnormal secretion which has become customary.* Relying especially on this symptom, Simon extirpated a calculous kidney. The autopsy afterwards showed that the other kidney was sound. This means of diagnosis will, of course, always leave us in the lurch whenever the retained calculus does not entirely close the ureter and the secretion of the diseased kidney can flow down alongside of it. Another evidence in favor of the calculous disease being unilateral, which must not be under-estimated, lies in the fact of the calculus being composed of phosphates; for, as we have already seen, this fact justifies the conclusion that the development of calculus is due to a purely local cause, whereas the observation of arthritic cases shows that the development of uric acid concretions very often has its foundation in constitutional causes, which are by no means limited to one kidney. Finally, we must remember the difficulties that attend the diagnosis in case of nephrolithiasis of a horse-shoe kidney, in which only one renal pelvis or one horn of the kidney is diseased, and, the ureter of the diseased side being obstructed by a stone, the other side produces normal urine.

If, as the result of a purulent, calculous pyelonephritis, a tumor is developed in the region of the kidney, after it has been determined to belong to the kidney, the composition of the urine will be of special value in settling the diagnosis. Piorry claims, in such cases, where a number of stones lie alongside of one another in the kidney, to have been able, by bimanual manipulation, to recognize the sound of the stones rubbing against one another.[1] The possibility of such a thing cannot, of course, be denied, but the conditions favorable to the production of this physical sign are doubtless very rare.

Duration, Terminations, and Prognosis.

Nephrolithiasis is ordinarily a very chronic affection. It is comparatively rare that any acute process caused thereby induces the fatal termination. This occurs when, as the result of the arrest of a renal stone during its passage through the ureter, a rupture of the latter takes place, whereupon sudden peritonitis quickly destroys life; or when, as the result of the complete arrest of the urinary secretion by obstruction of the channels of escape, uræmia is developed, which usually causes death within a few days with coma and convulsions. Those cases in which hydronephrosis or suppuration of the renal parenchyma takes place assume a very chronic course whenever the other kidney assumes vicarious functions—which it generally does—so that the duration of the disease is often protracted for more than a decade.

---

[1] Compare *Mayer*, Perc. des Unterleibes. 1839.

The *terminations* of nephrolithiasis may be:

1st, *Recovery.* This occurs in a series of cases in which only gravel or smaller concretions may be developed which can pass the ureter, and having reached the bladder are expelled thence with the urine; and in which, either spontaneously or as the result of appropriate treatment, a gradual diminution and final cessation of the development of renal sand and gravel is observed. But if more extensive stones have been formed which cannot pass the urinary canals, this method of recovery is not to be expected. Internal means prove utterly ineffectual for the dissolving of larger renal concretions. But in such cases, too, recovery has now and then been observed, even when destruction of the kidney had taken place by hydronephrosis or pyelonephritis. For, if the fluid has emptied itself, and is not produced anew, a shrinking of the diseased kidney may take place, the wasted organ causes no further disturbance, and if the other kidney is healthy, and provides for the secretion of urine, life may continue undisturbed. During the past year I have witnessed this termination of nephrolithiasis twice in making post-mortem examinations of persons who had died of other affections.

2d, *Death.* We have seen above that the fatal termination is seldom brought about by acute, but more frequently by a series of chronic processes which result from the presence of concretions in the renal parenchyma, the renal pelves, and the ureters. Furthermore, in the course of nephrolithiasis, a series of complicating processes (such as amyloid degeneration) are developed, partly in the other kidney alone, partly in this and the other abdominal organs, which finally induce a fatal termination.

The *prognosis* is evident from what has just been said. The earlier the disease comes under treatment, the better, in general, is the prognosis. In a certain series of cases it is possible to effect a diminution in the development of new sand and gravel, which might give occasion to the formation of larger concretions; and the concretions which exist, if they are not too large, may be floated out of the kidney by appropriate means. Still, even here we must not build up too great hopes. So-called cures are frequently but temporary improvements, and after a pause of years the former troubles may return. Besides this, we often find constitutional disturbances present whose removal may be quite impossible. If renal stones have existed for a long time, if they have caused resulting disturbances in the renal tissue, then the prognosis is much more unfavorable. For the favorable terminations which do, nevertheless, take place in individual cases are too rare to affect the general prognosis favorably.

Treatment.

The treatment of nephrolithiasis must have two objects in view, namely: 1. *To prevent the formation of renal sand and gravel, and to remove the already formed precipitates from the body* (the *radical* treatment); and 2. *To relieve and eventually to remove the symptoms and lesions caused by these concrements* (*symptomatic* treatment). In order to prevent the formation of the renal calculi, it is, in the first place, important to regulate the diet properly. Although there is still much dispute about some particular points, most physicians are, in general, convinced that a rich or exclusively flesh diet promotes the formation of renal sand.

The researches of Lehmann, and of Heinrich and Johannes Ranke, seem to prove beyond possibility of doubt that there is an increased excretion of uric acid, when the amount of animal food consumed is augmented. The fear of nitrogenized food, however, has been pushed too far. Lobb,[1] over a century ago, recommended an exclusively vegetable diet, and Magendie adopted a similar regime when he interdicted all nitrogenized nourishment. We know now that an abundant consumption of azotized substances is by no means the only cause of urinary sediments, although it greatly favors their production, particularly under certain circumstances. It is sufficient, then, to prohibit too excessive a use of meat, to recommend the use of white meat, and to insist particularly on great moderation in eating, and on the use of easily digestible articles of food which do not excite digestive disturbances. At all events, a suitable diet is one of the most important means of diminishing the excessive production of uric acid. An exclusively milk and vegetable diet is only to be recommended for persons in whom, in consequence of luxurious living, the excess of uric acid is very considerable. We must recollect, however, that an abundant consumption of nitrogenized food often does no harm when there is a corresponding consumption of the material of the body, such as takes place when the mode of life is active, and especially when much bodily exercise is taken. Vigorous individuals accustomed to high living must take constant exercise in the open air, in order to ward off bodily as well as mental inertia. Light out-of-door employments, such as gardening and riding, should be recommended. Fiery wines and highly seasoned foods must be especially proscribed. In order to remove already existing renal sand, the consumption of large quantities of fluid to increase the amount of urine has been recommended from ancient times. The fluid not only dilutes the urine, and diminishes the irritation of the kidneys and the urinary passages, and retains the solid matters in general of the urine in solution, but it enables the constantly forming precipitates to be more easily washed out of the kidneys and the renal pelvis. As a supplementary measure it possesses a decided therapeutic value. Of the simple therapeutic measures the most important is the abundant use of water. Spring water is recommended by most authorities, but a few prefer river water on account of its greater chemical purity.

---

[1] Treatise on Dissolution of a Stone. London. 1739.

Of late years ordinary water has been generally used only as an article of diet, it having been supplanted as a medicament by waters that contain soda, because the carbonate of soda accelerates the action of the fluid, and renders the use of such large quantities unnecessary, and because the carbonic acid supplies a healthy stimulus to the stomach. The action of the soda waters upon the uric acid precipitates will be described later. Ségalas recommended for the same purpose beer, which had already found an enthusiastic advocate in Sydenham. The latter tried it upon his own person, in order to dilute and cool off the ardent humors which remain in the kidney and produce the stone. By other authorities it is claimed that beer favors the formation of calculi. The beer of former times was, it is true, very different from that at present in use. At all events bad, sour beer is apt to excite catarrh of the urinary passages, and to aggravate already existing inflammation. In earlier times the most different diuretic drinks and teas were also recommended. How rich the therapy of the ancients was in this respect is shown by the list given by Joh. Varandaeus.[1] The following medicaments were then, and are in part still, considered to possess marked diuretic powers: dog rose-seeds, root of Cardana, rest-harrow, juniper and Pareira brava, uva ursi leaves, wall pellitory herb, etc. Usually combinations of different herbs of this class were employed. Decoctions or infusions of these herbs were taken with the addition of a few drops of nitric acid or muriatic ether. More powerful diuretics were also recommended, and they may be employed with advantage in some few cases where there is no irritation of the urinary passages. Among these may be mentioned cubebs, turpentine, and copaiba. It is necessary, however, to be very cautious in their employment, as they may aggravate an existing pyelitis, and increase the pain. Even coffee, which is interdicted in nephrolithiasis by most physicians, has found some advocates. In Italy, Palmieri's drops, which consist of washed sulphur and tar water, are much used for gravel. However, the employment of these, as well as of many other internal remedies recommended for this affection, can be justified neither on rational nor on empirical grounds. The same may be said of the employment of diaphoresis, which was strongly recommended by Civiale and others. *A priori* it must be conceded that the diminution of the amount of urine which is thereby induced renders the discharge of the urinary deposits more difficult, and that it therefore does more harm than good. The supposition that, when uric acid is copiously precipitated, a part of the acid may be excreted through the skin, requires positive proof.

On the other hand, lukewarm baths, and especially brine baths, are of undoubted value in nephrolithiasis, on account of the increased activity of the nutritive changes induced by them. They should be used as aids to other modes of treatment.

---

[1] De affectibus renum. Hanoviæ. 1617. p. 65.

Keeping the bowels open was recommended by Sydenham, who had personal experience of its benefits, and it is advisable to give a cathartic frequently, even when actual constipation does not exist. Sydenham took a cathartic (manna and lemon juice) upon a fixed day in every week for several months in succession, and states that it always gave him relief. Blood-letting, which Civiale recommended as a prophylactic against renal calculus, has found no advocates in practice. As a measure of symptomatic treatment it often proves useful in renal colic (see below).

Apart from these measures, there are a number of medicinal remedies which experience, in spite of the contradictions of a few prominent observers, has proved to possess the power of preventing the separation of certain urinary elements within the organism, and even of causing the solution of already formed sand, and perhaps of small grains of gravel. Larger concrements are not dissolved by these means. Every attempt to dissolve them chemically has as yet been futile. These remedies differ according to the different chemical constitution of the concretions, and an accurate diagnosis must in every individual case precede their employment. The first thing which must be required of these chemical solvents in order to obtain favorable results is that they be able to reach the kidney unaltered—they must be so-called *urophanic* substances.

The *alkalies* and *alkaline salts* are the solvents for *one class of renal sand.* This class comprises the sand and gravel which are composed of uric acid, urates of ammonia, cystine, and oxalate of lime. The alkaline treatment has also been employed for fibrinous concretions. The treatment of the uric acid deposits is the most important, because they are by far the most frequently met with. Of the remedies at our command, the mineral salts first come under consideration. Heller particularly recommends phosphate of soda in doses of from one to six drachms. According to him, the great advantage of this preparation is that, even when given in large doses, it does not excite diarrhœa, but the greater part of it is excreted in the urine. The presence of large quantities of the drug in the urine is absolutely necessary for the full development of its action as a solvent of uric acid. The phosphate of soda irritates the intestinal canal less than the carbonate of soda, of which we will speak presently. Finally, its action on uric acid is of importance, as it is able to hold in solution somewhat more of this acid than many other salts (Binz). In spite of all this, the preparation has not as yet been generally adopted in the treatment of nephrolithiasis. The value of the sulphates must not be underestimated, although in this connection they are only employed in the form of *water containing Glauber's salts* (vid. p. 735). The carbonate of lithia has also been recommended as a solvent of the uric acid gravel. The recent communications of Garrod[1] on the action of lithia require confirmation.

In addition to the above, alkalies in combination with carbonic acid and with the vegetable acids, which, as Woehler has shown, are excreted in the form of carbonates in the urine, may also be used. The carbonates particularly are very frequently used. Beneke recommends carbonate of potash instead of the favorite bicarbonate of soda, which, when largely used, causes, in his opinion, a diminution of the secretion of bile and disturbances in the production of blood-corpuscles. Alkalies are excellent as palliatives. They diminish the acidity of the urine, and in this way prevent the precipitation of the urates and of pure uric acid. Experience has taught us that the use of the natural mineral waters at the springs, *e. g.*, Vichy, Bilin, Salzbrunn, Neuenahr, etc., in combination with baths of the same waters, is far more efficacious than the simple medicinal use of the carbonates, or of the vegetable acids in the form of the juices of fruits, or even than drinking the alkaline waters at home. During the use of the mineral waters at the springs we frequently find that the precipitation of sediments ceases, and even permanent recovery may occur. These favorable results must not be ascribed solely to the purely palliative action of the alkalies, but are due in part to all the other factors which contribute so much to the efficiency of a course of treatment at the mineral springs—the entire change of diet and mode of life, the abundant consumption of water, the improved condition of the skin caused by the baths, etc. The baths not only, like the alkalies, aid in retaining the uric acid in solution, but they actually diminish its production.

---

[1] **Med. Times, March 22, 1873.**

The use of alkalies must in all cases be intelligently supervised, or, instead of benefiting, it will prove injurious. In the first place, they must not be given in such large quantities as to give the urine an alkaline reaction. If this should occur, it is certainly very probable that the urates and uric acid will no longer be precipitated ; but, on the other hand, a precipitation of the earthy phosphates from the urine within the urinary passages will unavoidably occur. In this way phosphatic concretions are formed ; or, when other varieties of concrements already exist—*e. g.*, in the renal pelvis—the phosphates are deposited upon their surfaces, and enlarge them by incrustation. The waters that contain Glauber's salts, in the first rank of which must be placed those of Carlsbad and also those of Tarasp, possess a marked superiority over the pure soda waters. They act more certainly and reliably—so much so, that the waters of Carlsbad, though they contain only a little over a third as much soda as those of Vichy, nevertheless compete successfully with them. Whether the greater activity of the former be due to the presence of the sulphates, or to the high temperature of the water, or to the quantitative combinations of the salts, cannot as yet be decided ; the fact, however, is undeniable. Seegen's investigations have proved that the water from the Carlsbad mill-spring, which contains chiefly sulphate of soda, common salt, and carbonate of soda, will cause a distinct decrease, and even the disappearance, of the uric acid. From the time of Hufeland the springs of Wildungen have enjoyed a great reputation, but they are far less powerful than those mentioned above. They may answer when the use of the waters can be continued for a long time, and as an adjunct to the stronger waters. Their active ingredients are principally bicarbonate of lime and bicarbonate of magnesia. Thompson, of London, who has had a large experience with calculous diseases, has recently [1] recommended a somewhat complicated course of treatment, by which he claims to have been especially successful in preventing the excessive production of uric acid and the formation of renal concretions.

---

[1] **Lancet. 1872. 13. January.**

He orders, in the first place, from two to three grains of blue pill to be taken at night, and the next morning, from seven and and a half to nine and a half ounces of the natural Friedrichshall bitter water; then for one to three weeks he gives the same bitter water, with warm water, in diminishing doses, because when used for a long time it produces the same effects in smaller doses. After this, he gives for three weeks a combination of natural Friedrichshall water, four ounces; Carlsbad water, from five to six ounces; and hot water, from three to four ounces. The course of treatment is closed by the use, for two weeks, of from six to seven and a half ounces of the artificial Carlsbad water. The treatment lasts altogether six to eight weeks. Alcoholic drinks, fatty and saccharine foods, are not allowed during this time. On the other hand, suitable exercise in the open air is necessary. The vegetable alkalies (especially in the form of the grape-cure) may be cautiously used as an after-treatment, provided they are not otherwise counter-indicated. Those juices must be avoided, however, which contain oxalic acid, for fear of promoting the development of concretions of oxalate of lime. The treatment of the *oxalic acid gravel* must be conducted on the same principles as that of the uric acid precipitates. As to the treatment of *cystine gravel*, we must admit that we know of no remedy which prevents its formation. The cystine is dissolved, it is true, by alkalies, but we must bear in mind that the formation of phosphatic concretions occurs very rapidly in alkaline urine.

The value of alkalies when *fibrinous concretions* are present depends on the fact that they are said to facilitate the solution, and consequently the discharge, of the concretions.

The employment of acids is frequently recommended in the treatment of the *second* class of renal sand and gravel, to which belong, in addition to the ammoniaco-magnesian phosphate, the phosphate of lime, the carbonate of lime, and a mixture of the earthy phosphates and carbonate of lime. The mineral acids, which are much used in England, especially muriatic acid, are useless, because, to enable them to pass unchanged into the urine, they would have to be given in poisonous doses. The only urophanic acid is carbonic acid (Heller), to the therapeutical importance of which in this connection even Mascagni, but particularly Thénard, called attention. It may be administered in the form of carbonic acid water or of vegetable acids (acetic, vinous, citric, malic acids, etc.), which are changed in the body into carbonic acid and water. Heller states that he has observed in several of his patients that the urine, which was usually cloudy, on account of the bone-earths it contained, and often even contained a large quantity of sediment, became perfectly clear whenever carbonic acid water was drunk. In some few cases, indeed, fragments of concretions were passed as sand. While recognizing the value of this experience, we must avoid indulging in illusive hopes of cure from the treatment, because we know that the pathogenesis of these concretions finds its explanation principally, if not exclusively, in local causes, namely, a catarrh of the mucous membrane of the renal pelvis, with subsequent ammoniacal decomposition of the urine. So long as this continues, a cure by chemical means cannot be expected.

If we do not succeed by dissolving the renal sand and gravel within the body, or in preventing the formation of larger renal concretions, we must direct our attention to moderating the suffering caused by their passage through the urinary canals, and to treating symptomatically the changes produced by them in the urinary organs. And first, as to the treatment of renal colic. The causal indication is to relieve, as rapidly as possible, the retention of urine induced by the impaction of the calculus. "Cessante causa cessat effectus." Simpson,[1] in two cases, employed successfully for this purpose a measure that was as simple as it was ingenious. He caused the patients to be held head downward, and at the same time rubbed the affected side. The concretions changed their position, and fell back into the distended renal pelvis, and the discharge of the urine was again established. As a rule, however, the attack cannot be cut short in this manner. We are then restricted to a purely symptomatic treatment. The patient must keep as quiet as possible. Narcotics give the greatest relief. General blood-letting is too uncertain and too severe a measure for most cases, and is at most only indicated in plethoric individuals with active congestion. Local extractions of blood are applicable when there are violent local inflammatory symptoms. The doses in which the narcotics may be given vary according to the severity of the attack and the idiosyncrasies of the patient. Opium and morphia alone come into question here. If the pains moderate, or narcosis sets in, the remedy must either be entirely suspended or given in smaller and less frequent doses. If vomiting should prevent the internal administration of these narcotics, the subcutaneous injection of morphia may be employed—a mode of administration which is, moreover, often preferable from the start to the internal use of the drug. The administration of opium in an enema (from ten to fifteen drops of laudanum in a wineglassful of thin starch) often gives good results. Narcotic cataplasms are also often useful. In a few cases cold applications give better results. The usefulness of chloral must be borne in mind. Sometimes the combined action of chloral and morphia produces a prolonged euphoria, which neither of these remedies alone is able to do. In very severe cases inhalations of chloroform must be cautiously administered in order to induce at least temporary rest. The external application of chloroform accomplishes just as little as other cutaneous irritants. The internal administration of the anti-spasmodics is also utterly useless; camphor alone having found favor with a few trustworthy authorities. When the attacks are of long duration, protracted lukewarm baths may be employed, and also remedies which increase the secretion of the urine, in order to increase the *vis a tergo* and force the stone onward. Emetics or other forcible remedies are much too unreliable to deserve consideration here. If the symptoms of a pyelitis or a pyelonephritis caused by the concrements be present, the treatment described in the chapter on those affections must be employed.

---

[1] Edinb. Med. Journ. 1868.

If the evidences of a perinephritis, which have been fully described in the appropriate chapter, be developed, we must hasten, in order to prevent the burrowing of pus and perforation into other organs, to lay open the abscess, and at the same time perform the operation which has been long known under the name of nephrotomy. Troja defines nephrotomy as an operation in which a deep incision is made in the lumbar region, extending into the kidney or the renal pelvis, in order to remove a calculus situated in these parts. He considers it to be settled, however, that the operation should not be performed unless there be a pre-existing abscess, or the signs of a tumor. This variety of nephrotomy is that which is still practiced. Dr. Dawson[1] had under treatment a man with a large tumor in the left lumbar region, in whom he diagnosed pyelitis and a renal calculus. He drew out a pint of pus through the canula of an aspirator, and five days later made an incision over the tumor 7 ctms. (2¾ in.) deep, evacuated the pus with a trocar, enlarged the opening, and removed the stone with his hand. The wound was drained. Pyæmia set in on the fourth day after the operation. It seems that this operation has never been performed where there has not been a demonstrable tumor. Five cases of this sort, it is true, were already recorded in Troja's time, but he regarded all of them as insufficiently authenticated. The best known case of the kind was reported by the English consul at Venice. The operation was performed by Dominicus de Marchettis at the end of the seventeenth century. In modern times this operation has been frequently condemned by surgeons, and Malgaigne insisted that it should never be allowed to pass out of the anatomical amphitheatre into surgical practice. . The extirpation of the kidney for the cure of nephrolithiasis was never seriously thought of before Simon recommended it (1871). Troja mentioned it as an extraordinary and ridiculous sort of nephrotomy. Rayer considered the extirpation of a kidney that contains a calculus impracticable, on account of the adhesions which were invariably present. The most recent authors also reject this operation, because a positive diagnosis of renal calculus, especially when unilateral, seems to them impossible. This explicit rejection of the operation is based upon the experience of Durham and Gunn (1870), who cut down into the renal pelvis to remove calculi; but when they got so far that they could palpate the kidneys without any tissues intervening, they found no stones and had to abandon the operation. Simon also met with a case of this sort. He advises, in order to remove all doubt, that after exposing the kidney, and before deciding upon extirpation, acupuncture should be practised with fine, long needles, by means of which the presence of a calculus can be readily ascertained. Simon was the first to extirpate the kidney for nephrolithiasis. Although his patient died of septicæmia on the thirty-first day after the operation, still the death was not due to the nature or the severity of the operation, but to an accidental complication.

---

[1] Schmidt's Jahrbb. Bd. CLVII. S. 70.

The description of the details of the operation belongs to the domain of surgery. Simon's works contain the most minute instructions for it. As to the justification of the operation, as little can be said against it as against ovariotomy. In nephrolithiasis, as in ovarian tumor, there is a certain stage of the disease when all other methods of treatment are useless, and both, sooner or later, inevitably lead to a fatal termination. Naturally the diagnosis of the calculous affection of the kidney in general, and of the involvement of only one kidney, must be sufficiently positive (comp. p. 727) before such a severe operation is decided on. The exploratory puncture recommended by Simon after the kidney is exposed would be advisable in every case. Many more cases must be collated before a definite judgment can be formed upon the subject. At any rate, Simon's procedure deserves imitation in analagous cases. Even if errors in diagnosis should be made in a few cases, there would be as little justification for condemning the operation of nephrotomy on that account as there would be for discarding ovariotomy, which is to-day universally recognized as a most useful operation.

According to Simon, the extirpation of the kidney should be performed when the greater part of the substance of the organ is still in a state of preservation, while he recommends the laying open of the kidney and removal of the stone (the operation which has till now been called nephrotomy) when the retention of the urine has caused such extensive destruction of the renal substance that it forms only a sac, an incision into which causes no bleeding. In such cases the renal wound must be kept open, and an attempt must be made to make the sac to heal up from the bottom. Not infrequently it will be necessary to keep the renal fistula open for a long time. Otherwise a retention of pus might easily occur, which, apart from its other evil effects, might give rise to new renal calculi.

The Animal Parasites of the Kidneys.

The Echinococci of the Kidneys.

Literature and History.

In addition to the authorities mentioned on page 543:

Bremser, Lebende Würmer im leb. Menschen. Wien. 1819.—*Leuckart*, Menschliche Parasiten. I. Leipzig. 1863. S. 335.—*Davaine*, Traité des entozoaires et des maladies vermineuses. Paris. 1860. p. 524.—*Béraud*, Des hyatides des reins. Thèse. Paris. 1861.—*Schmidt's* Jahrbb. CXLIV. S. 52. u. CLII. S. 96. The reports of cases scattered in various periodicals.

Although Pallas, as early as 1760, described the echinococci as independent parasites, and admitted a certain relationship between them and tænia—observations which were confirmed by the naturalist Pastor Goetze—yet a long period intervened before these views were generally accepted by the great medical public. A few observers almost discovered the true nature of these echinococcus tumors, but on the other hand the most different varieties of renal cysts were frequently confounded with them. Baillie, it is true, described them unmistakably as early as the commencement of this century, under the name of "genuine cysts,"[1] as cysts with a capsule which contains a number of hydatids. The descriptions given by him are perfectly true to nature. The hydatids, says Baillie, have white, half opaque membranes, like the hydatids of the liver, contain a fluid coagulable by acids (this is, however, only exceptionally the case when suppuration of the capsule has taken place), and have the power to produce smaller vesicles. The smaller hydatids are sometimes fastened to the membranes of a larger one, sometimes float free in its cavity. In describing the analogous hepatic cysts he expresses the opinion—somewhat reservedly, it is true—that the animals contained in these cysts possess a very simple structure. Notwithstanding Baillie's description, Koenig (1826) frequently confounded the echinococcus cysts of the kidney with serous cysts.

---

[1] Engravings. VI. Fasc. plate VII.

Bremser furnished the first exhaustive description of the echinococci of man. Although the animal nature of these parasites was known to him, he regarded it as possible that external violence might excite their development. The natural history of the parasites was more closely investigated in more recent times by von Siebold, Kuechenmeister, van Beneden, and Naunyn. C. Davaine makes use of the existing material, in a masterly manner, in his excellent work. Since then a pretty large amount of clinical material has accumulated in the professional journals.

Etiology.

The echinococci are the most frequent animal parasites of the kidney in our latitudes. They are the tæniæ at one period of their development, and represent the immature condition of a tape-worm that inhabits the intestine of the dog, the tænia echinococcus. The brood of this intestinal worm enters under certain circumstances the human digestive canal, and thence the parasite makes its way into the different organs, and there gives rise to the development of the echinococcus cysts. The more or less frequent occurrence of the latter will stand in direct ratio to the number of dogs affected with the tænia echinococcus, and the more or less intimate intercourse between single individuals, or the population of entire districts, and the dog. Reliable observations upon this point are few in number. In England, France, and Germany, the echinococci are not rare; they are more rare in India and America. According to Whyttel, physician to the Adelaide Hospital at Melbourne, the echinococcus is much more frequent in South Australia than in England, and it seldom happens that there is no case of it in the hospital. In Egypt, Bilharz found a similar state of affairs. The echinococcus, however, is met with most frequently in Iceland, according to all the observations as yet obtained. A seventh, and even according to some authorities a fifth part of all the deaths on that island are caused by echinococci. The echinococcus is also very frequent in Mecklenburg[1]—in 251 autopsies it was found twelve times (1 : 21). In Silesia it is not rare ; in Breslau, especially, it was found thirteen times in 2,006 autopsies.[2] According to the experience of Frerichs, they are found far more frequently in Silesia than in Göttingen, Kiel, and Berlin.

The causes of the excessive frequency of echinococci in Iceland are to be sought not alone in the great number of dogs that exist there, but also in the facts that these dogs exceedingly often harbor the tænia echinococcus, and besides live in extremely close intercourse with the natives. Without examining more closely the social possibilities of transplantation, it is evident that the opportunities for it are certainly amply supplied during the long, close cohabitation of man and dog during the northern winter. In Mecklenburg the frequency of the echinococci is, according to Wolff, dependent upon the great number of canine tæniæ. With regard to Silesia, in particular, Lebert[3] attributes the frequency of echinococci, undoubtedly with reason, to the consumption of dog's meat by the poorer classes. It is certainly probable enough that during the different manipulations of dog-butchering the eggs of tænia should sometimes escape from the intestines and fasten on the flesh.

With regard to the seat of the echinococcus cysts in the human organs, they are met with in the kidneys, according to the comprehensive statistics of Davaine, somewhat less frequently than in the lungs, and twelve times less frequently than in the liver, but more frequently than in the remaining organs. The cause of the kidney being relatively less frequently involved is to be found in the fact that the young animals can find their way out of the stomach most rapidly and easily into the liver. Of the reasons why in other cases, however, they wander directly into the kidneys, and at the same time often leave all the other organs free, we are unable to form even a supposition. With regard to the remaining etiological influences, the statement that men are far more frequently affected with echinococci of the kidneys than women seems to be well founded. Roberts collected sixty-three cases, of which forty-one were men and twenty-two women. In Iceland, however, both sexes are affected in pretty equal proportions. The echinococci are developed most frequently during the prime of life, between the ages of twenty and forty years; in Iceland, between the thirtieth and the fiftieth year. However, isolated cases are also met with in infancy and in advanced age.

---

[1] *Wolff*, Berl. klin. Wochenschrift. 1870. Nr. 5 u. 6.

[2] *Wolff*, Inaugural Dissertation. 1869.

[3] Dissertation von *Schmalfuss*. 1868.

Pathology.

Pathological Anatomy.

The echinococci are rather more frequently found in the left kidney than in the right, and very rarely in both kidneys. In a few cases they have been found in the liver or in other organs, as well as in the kidney. Usually the parasite is seated in the renal tissue, rarely between the capsule and the gland. As the echinococcus grows the renal substance atrophies in a similar ratio, so that finally the entire organ may be destroyed. The outer covering of the echinococcus then consists of atrophic and anæmic renal substance, which in some places has even been entirely destroyed. Often, however, in what remains of the parenchyma the uriniferous tubules will be found still well preserved, along with a more or less marked increase of the interstitial connective tissue.

The echinococcus cysts of the kidney form rounded, fluctuating tumors, varying in size from an egg to a gourd. They project considerably above the surface of the kidney. The echinococcus cysts have a great tendency to perforate into the renal pelvis. The not very numerous post-mortem examinations prove that the cysts open into the renal pelvis most quickly when they are developed in the medullary cones. The tumor in these cases does not attain a large size. When the parasites are developed in the cortex, or between the renal capsule and kidney, the cysts attain the largest dimensions of which they are capable. When a rupture into the renal pelvis has taken place, the anatomical appearances of pyelitis will be found. The cysts may also perforate in other directions, *e. g.*, into the bronchi. A rupture into the peritoneum has, as yet, never been observed. In the vicinity of such a cyst more or less extensive adhesive peritonitis is often developed. The echinococcus is enclosed by a firm, fibrinous, white or yellowish capsule, which is intimately united with the surrounding glandular tissue and is highly vascular. Its thickness varies between 1 or 2 mm. ($\frac{1}{20}$ to $\frac{1}{10}$ in.) and 0.75 to 1 ctm. ($\frac{3}{10}$ to $\frac{2}{5}$ in.). Completely filling this connective tissue covering, the inner surface of which is smooth, there is a gelatinous, translucent sac consisting of numerous concentric hyaline layers, the so-called mother vesicle of the echinococcus. This sac contains a clear, watery fluid, in which usually numerous large and small vesicles move about freely. Some of these, especially the smallest, are still attached to the mother vesicle. The size of the vesicles varies from a millet-seed to a goose-egg. The large vesicles sometimes contain smaller ones of a third generation, and, now and then, even a fourth generation is found. The growth of the mother vesicle is proportionate to the increase in the amount of the fluid and the number of daughter vesicles. The individual scolices are developed on the inner wall of the vesicles in the form of delicate white nodules as large as a millet-seed or smaller; as a rule, they are found in groups, but are sometimes swimming about in the fluid. A closer examination shows that the parasites possess a tapeworm-like head, having four suckers and a proboscis. The latter is surrounded by a double row of hooklets, the number of which, according to Kuechenmeister, varies between 28 to 36 and 46 to 52. The appearance of the animal varies greatly, according as its head is stretched out or drawn in. The head of the worm is separated from the body by a furrow, and on the posterior surface of the body there is a depression resembling a navel. At this spot the pedicle is inserted, by means of which the animal is fastened to the inner surface of the vesicle. Whyttel observed rapid movements at the point of union of this cord with the germinal membrane, which were caused by the vibrating cilia-like processes on the funiculus. These movements continued until the echinococcus died. The body presents longitudinal striations which run from the head backward, and also transverse striations. In addition to the striæ more or less numerous calcareous bodies are found in the animal. These parasites can live for a long time suspended in the fluid of the vesicles, when removed from the human organism. Waldeyer found them still able to move on the second day after their removal from an echinococcus cyst of the kidney.[1] In other cases the daughter vesicles are entirely absent and only one large sac is found, upon the inner surface of which the echinococci are seated, or it may contain no echinococci at all. This is the variety of echinococcus which has been repeatedly described (by Davaine and others) as a simple developmental stage of the hydatid, and which was described by Laënnec under the name of acephalocysts, and by Kuechenmeister as sterile echinococcus. In all these cases the echinococcus sac is of considerable size.

The membrane of the echinococcus presents frequently an exceedingly fine lamination, and does not consist of proteine. Luecke's opinion that they contain chitine, which, when treated with sulphuric acid, yields grape sugar, seems to be well founded. The fluid in the vesicle is colorless, clear, or slightly opalescent, usually of neutral, now and then of alkaline or acid reaction, and contains no trace of albumen. Its solid contents consist principally of inorganic substances, and especially of ordinary salt. Heintz and Boedeker found succinate of soda in it ; Naunyn found inosite in the echinococci of animals, and Wyss found it also in those of men. Large quantities of chloresterine crystals are also found in this fluid, as well as oftentimes hæmatoidine crystals. A large amount of albumen is found in it after puncture of the sac. In the fluid of renal echinococci, crystals of uric acid, oxalate of lime, triple phosphates, and earthy elements also occur (Barker). The echinococcus cysts often form extensive adhesions with the neighboring organs. Suppurative processes may also be set up in their vicinity. The cysts themselves may suppurate or they may atrophy. In the latter case the echinococci and the membranes perish, the fluid is absorbed, and the sac contracts into a dense, firm mass. This mode of termination is met with when the contents of the cysts have been previously emptied by the rupture of the sac, but oftentimes also without previous rupture. These shrivelled-up echinococcus cysts contain a whitish, chalk-like, crumbling or greasy mass. Formerly these masses were often wrongly regarded as tuberculous products. The folded, compressed, easily recognizable membranes will frequently prevent this mistake being made even at the macroscopic examination, and in all cases the microscopic examination will clear up the diagnosis. These masses consist of amorphous phosphate of lime, crystals of the ammoniaco-magnesian phosphate, cholesterine tablets, fat-drops, and particularly echinococcus hooklets, as well as, finally, small shreds of laminated membranes.

---

[1] *Bufe*, Dissert. inaug. 1867. Breslau.

Symptomatology.

The symptoms produced by the renal echinococci are manifold, and often difficult to interpret. The only characteristic symptom is the discharge of individual parasites per urethram. So long as this does not occur, and the cyst remains intact, its course is latent until it has attained a sufficient size to be felt as a tumor in the lumbar region. Since usually only one kidney is the seat of the echinococci, and the healthy kidney secretes vicariously, the parasite may exist for a long time without causing any disturbances in the renal secretion.

When the echinococcus sac attains large dimensions it pushes aside the neighboring organs, as a rule, without interfering with their functions. The positions of the tumors produced by the echinococci, and their relations to the neighboring organs, are, in general, the same as those of other renal tumors, which have been fully described, particularly in the chapter on renal cancer. If these tumors lie directly under the abdominal wall, they are dull on percussion; if the colon lies between them, the percussion note is dully tympanitic. When the tumor is seated in the right kidney, and is developed in the upper portion of the organ, it is in contact with the liver, and as the line of demarcation between the two cannot be made out, it is often mistaken for a hepatic tumor. When the echinococcus sac is developed on the left side and upward, it may extend as high as the spleen. When the tumor develops downward, it may extend as far downward as the iliac fossa. When it attains such an immense size it may become troublesome to the patient in consequence of the feeling of pressure, weight, and tension caused by it. The renal echinococci, which form such distinct superficial tumors, are much exposed to external injuries, such as blows, falls, etc. Inflammation of the cysts, or rupture of the same, may very often be traced back to such traumatic influences. Only about half the cases of renal echinococci produce a tumor. The tumor is usually about as large as an orange or a child's head, and on palpation it is found to be round, and apparently tensely distended. In a few cases the fluctuation is demonstrable; in other cases it is indistinct.

The so-called *hydatid fremitus*, which is regarded as a particularly valuable diagnostic sign, to the semiotic importance of which Piorry and Briançon especially drew attention, can only be obtained in a small number of the cases. It will be felt most distinctly when the tumor is lightly compressed by two fingers of the left hand, and a slight tap is given to it with the right hand, or when the finger is allowed to rest for a time upon the pleximeter after the percussion stroke. A similar sensation will be imparted to the auscultating ear by lightly tapping with the finger over the corresponding part of the abdomen. Frerichs, in examining hepatic echinococci, could only obtain the hydatid fremitus when the echinococcus sac enclosed a great number of vesicles, and was not very tensely distended. When only one distended bladder existed, he could not produce the sign. Other observers have also obtained the hydatid fremitus even under these circumstances. The experiences that have been communicated upon this point with regard to renal cysts show that the sign may be absent even when the most favorable conditions for its production are present.

When the cysts burst, and their contents empty into the renal pelvis, as they do in fully two-thirds of the cases, a characteristic group of symptoms appears. These are often, but not necessarily, complicated symptoms, due to affections of the kidney. Pains in the renal tumor and along the ureter are often observed. The most characteristic sign, however, is the discharge of entire echinococcus vesicles and fragments of vesicles with the urine. Sometimes these alone are discharged, but at other times the urine also contains a whitish, milky detritus, in which echinococcus hooklets or shreds of laminated membranes may be found. The discharge of these vesicles and fragments occurs in attacks, during which the cyst apparently becomes entirely emptied, and finally shrinks up. In the majority of cases, however, this does not take place so rapidly. Usually several such attacks follow one another, the free intervals lasting several days, several weeks, months, or even years. These attacks usually begin with violent pains in the hip ; the patients often have a feeling as if something had burst internally. The pain shoots downward along the ureter to the inner side of the thigh. It may be accompanied by a chill, nausea, and singultus. This, however, is rare. Colicky pains in the course of the ureter next set in, and warrant the conclusion that the vesicles are passing through the ureter. Sometimes the attacks take on a more serious character in consequence of the retention of the urine. In the severe attacks the testicle is retracted, as in the renal colic caused by the passage of a calculus. These symptoms last from a few hours to several days, and they usually cease suddenly, with a sensation as if something had fallen into the bladder. A new set of symptoms consequent on the retention of the urine in the bladder will now be excited: severe strangury, with pains which radiate into the head of the penis. The evacuation of the bladder is followed by immediate relief. Sometimes the vesicles, which obstruct the urethral opening, stick fast in the eyes of the catheter during the attempt to draw off the urine accumulated in the bladder, and are discovered when the instrument is withdrawn. This discovery of the corpora delicti furnishes an infallible guide to the diagnosis.

Women have been seen to draw out with their own fingers the vesicles which block up the urethra. The number of vesicles discharged during an attack varies from one or two up to several dozen. These either float in the urine, or are collapsed, and are found in the sediment at the bottom of the glass.

The urine is often tinged with blood and mixed with pus, in consequence of a complicating pyelitis. Bloody urine is usually passed for a considerable time before the cyst ruptures. The urine is more or less dark colored, according to the quantity of blood it contains. If blood and pus be absent, the urine may be perfectly clear.

Blood-globules and pus-corpuscles are found in the sediment in variable quantities. When the urine is alkaline, crystals of the triple phosphates and of urate of ammonia make their appearance ; the hooklets of echinococci occur as a characteristic admixture. The exertion of strength employed in the expulsion of the vesicles is often sufficient to force them out of the urethra with a distinct noise and to carry them a certain distance. The paroxysms are occasionally excited by a distinct exciting cause, such as a blow or a fall, or by riding or driving. In one case the attacks usually followed the use of spirits or of strong coffee. In many cases the pains in the diseased kidney increase before the rupture of the tumor takes place. In a number of cases the tumor does not decrease in size after the rupture, or even after an abundant discharge of vesicles, but, on the contrary, it often increases in size. When, for instance, the vesicles become impacted while passing through the ureter, an acute hydronephrosis, with noticeable swelling of the tumor, often arises, which, if the hinderance to the discharge of the urine be not overcome, may become permanent, and produce a large renal tumor. When, however, the vesicles, which were wedged in the ureter and obstructed the discharge of the urine, are forced onward, or when no such obstruction occurs, a diminution in the size of the tumor is observed, which is in direct proportion to the quantity of vesicles voided.

In a few cases paresis of the lower extremity corresponding to the affected kidney has been observed. One of the cases observed by Frerichs is an instance of this.

The symptoms are different when the cysts empty themselves in other directions. These cases, however, are very rare. We may mention the simultaneous rupture into the renal pelvis and the bronchi. If in such a case a bronchus communicates with the renal pelvis, a distinct urinous odor, in addition to the characteristic membranes, and eventually the elements of the urine, can be detected in the expectoration. Sometimes the evidences of a perinephritis are present prior to such a perforation. At the present time no sufficiently accurate observations of perforations in other directions exist to enable us to present a reliable clinical picture of them.

Suppuration of the sac is accompanied by high fever and chills, and is usually excited by violent causes, such as blows, falls, etc.

Diagnosis.

The diagnosis of renal echinococci becomes certain under the following circumstances: 1. When a cystic tumor can be demonstrated which undoubtedly belongs to the kidney; 2. When echinococcus vesicles are discharged with the urine, with the symptoms of a nephritic colic; and 3. When, simultaneously with this discharge, the tumor in question diminishes in size. If the last symptom be wanting, the diagnosis cannot be made with absolute certainty, and can only possess a high degree of probability.

I had an opportunity to examine a case of this sort. A man, between thirty and forty years of age, presented a tumor, which could be referred with certainty to the left kidney. He handed me a bottle of urine in which a number of echinococcus vesicles floated about. He had been passing these vesicles for several days, their passage being accompanied by colicky pains, and they excited his interest all the more as he had previously heard the most diverse diagnoses of the nature of his malady. The urine was acid, bright yellow, and contained a little purulent sediment, in which the microscope revealed many echinococcus hooklets. Unfortunately, I have been able to learn nothing further of this patient, who was a shoemaker from the province of Silesia.

The simple discharge of echinococcus vesicles with the urine does not justify a positive diagnosis, for a few rare cases have been met with in which echinococcus cysts in the pelvis rupture either into the intestines, or into the bladder and the intestines together, or into the bladder alone. In the two last events the echinococcus vesicles pass off with the urine, although no demonstrable tumor in the kidneys exists. Then, too, the pains along the ureter, which precede the discharge of the echinococcus vesicles, are absent. The examination per rectum and per vaginam in such cases reveals usually the existence of the tumor in the pelvis, which furnishes the vesicles. Great caution is necessary in the interpretation of the symptoms, when echinococci are discharged through an abscess in the lumbar region. In spite of the suspicion which the locality excites, they do not by any means necessarily originate in the kidney. Cases have been observed and confirmed by the autopsies, in which under these circumstances the echinococcus tumors had been developed entirely in the muscles of the lumbar region. In addition to these difficulties in the determination of the organ in which an echinococcus tumor of the abdomen is located, the answer to the question whether a given cystic growth of the kidney be an echinococcus tumor or not, is often very difficult. The examination of the fluid drawn from the cyst can alone decide this question with certainty. The clearness, the low specific gravity, the absence of albumen, but, above all, the discovery of hooklets and laminated membranes, speak for echinococci. No positive reliance can be placed upon the presence or absence of the hydatid fremitus formerly regarded as so important. In the first place, it is exceedingly inconstant, and as Jobert found it in echinococcus cysts, which consisted of a single large sac, it is not at all improbable that it also occurs in other simple cystic tumors of the kidneys, and hydronephroses of large size. The differential diagnosis between echinococcus tumors and hydronephrosis is consequently not possible without explorative puncture.

Of especial practical importance is the differential diagnosis between *ovarian tumors* and *echinococcus tumors of the kidneys*, which may be mistaken for one another in consequence of the development of the latter downward into the cavity of the true pelvis—a mistake, which may have exceedingly dangerous consequences. The differential diagnosis between ovarian and renal tumors has already been considered at length in the chapter on Renal Cancer. Here a short clinical observation must suffice instead of further discussions.

Spiegelberg[1] operated on a woman at forty-two years, but, instead of the expected ovarian cyst, found a renal echinococcus. The tumor had been developing for one and a half years in the right hypogastrium, and had attained the size of a man's head. It was very elastic, irregular, very movable, and a slight murmur resembling the uterine souffle could be heard over it. A part of the tumor extended for a short distance into the anterior portion of the pelvic inlet. It was hard, painless, and situated close under the abdominal wall. The tumor could only be removed incompletely with a portion of the right kidney. The adhesions with the surrounding parts were firm and numerous. In this case, as Spiegelberg rightly states, the explorative punction would have been the only means by which the lamentable error could have been prevented. Only in that way could the possibility of its ovarian origin have been excluded. The hydatid fremitus was wanting in this case.

---

[1] Archiv f. Gynäk. I. S. 146. *Bufe*, Dissert. inaug. 1867. Breslau.

I have already mentioned above that the discharge of echinococcus vesicles with the urine does not in itself authorize a diagnosis of echinococcus of the kidney with rupture into the renal pelvis. On the other hand, however, it does not seem to me justifiable with our present knowledge of the course followed by echinococci of the liver, to assume a rupture of the latter into the bladder, as long as no anatomical proofs of it exist. In such cases it is far more likely that we have to deal with renal echinococci.

Schmalfuss has reported a case of this kind that occurred in Middeldorpff's Clinic.[1] The whole course of this case seemed to indicate that an echinococcus sac of the right kidney had penetrated into the renal pelvis. The patient, a laborer in his thirty-third year, previously healthy, had noticed for some time some emaciation of his body and swelling of his belly. He was received into the Clinic on account of sudden retention of urine. A dull percussion note, covering a space as broad as the hand, extends downward from the level of the normal hepatic dullness on the right of the navel, and is lost in the flat percussion sound over the greatly distended bladder. A very strong catheter brought away a whitish, quite characteristic echinococcus membrane, which was caught in its fenestra, and a slight spontaneous discharge of slightly bloody cloudy urine followed. Later on, more vesicles of the same sort, in which distinct scolices and hooklets could be demonstrated, were extracted in the same way. After this the patient discharged with the urine, which was of a bright yellow color and deposited a very purulent sediment, numerous echinococcus vesicles from the size of a lentil to that of a walnut. From that time forth he voided his urine freely and easily, and passed no more echinococcus vesicles. A moderately severe catarrh of the bladder was left behind. During the next two days the patient had fever without any other manifestations of disease. These slight febrile movements and pains in the belly continued for the next month, but he was able to return to his work. Finally these symptoms also disappeared. The dullness described above was permanent. The patient felt quite well.

---

[1] Inaugural-Dissertation. 1868. Breslau.

Duration, Course, Prognosis.

The duration is wholly indeterminable. Sometimes recovery sets in after a single discharge of vesicles with the urine. As, however, a fresh discharge of vesicles has been known to take place after an interval of ten years, it is almost useless to speak of a permanent cure. Neither the number of the discharged vesicles nor the frequency of the discharges furnishes here a sure clue. In general, all that can be said is that, when after one or more thorough evacuations no fresh ones follow for a long time, and the tumor diminishes in size so much that it can no longer be mapped out, the probability of the cure being permanent constantly increases with the lapse of time since the last discharge. Before the echinococcus tumors have burst, of course no opinion at all can be formed as to the duration of the affection.

The *course* is in general favorable when the echinococcus sac bursts into the renal pelvis; either recovery sets in after one or more discharges of the vesicles, or fresh discharges of echinococcus vesicles continue to take place at long intervals. *Death* may ensue in different ways: through rupture into the bronchi, through suppuration of the sac, etc. Further, a fatal result follows inevitably when, as was observed in a case recorded in Roberts' work upon the diseases of the kidneys, a solitary kidney is the seat of an echinococcus cyst. In that case the cyst burst into the renal pelvis. In addition to the echinococcus, a calculus was found. Death follows unavoidably and rapidly from the cessation of the function of the single kidney. In general, however, the prognosis is more favorable when the echinococcus is situated in the kidney than when it is situated in other organs. It is most favorable when the contents of the cyst are discharged into the pelvis renalis and thence outward through the urinary passages. Rupture of the cyst into other organs, *e. g.*, into the lungs, justifies an unfavorable prognosis. Experience teaches that the prognosis is worse when the cysts attain a great size, for two reasons: first, because large cysts impinge on the thoracic cavity and interfere with the functions of the vital organs situated there; and secondly, because they are more exposed to contusions, which are not unfrequently followed by suppuration of the cyst.

Treatment.

The first object of treatment is the prophylaxis. In a country like Iceland, where about every seventh person dies of echinococcus, it should be one of the principal aims of the sanitary police to prevent as far as possible the development of the parasites in man. The consideration of the means to be adopted for this end belongs to the treatment of the echinococcus disease in general (vid. Vol. III.).

With regard to the special treatment of echinococcus cysts of the kidney, the attempt to destroy the parasites by means of iodide of potassium and mercurial preparations has thus far been unsuccessful. We cannot even influence by medicaments the bursting of the cysts. When, however, perforation into the renal pelvis has taken place, the employment of mild diuretics to expedite the washing-out of the parasites through the urinary passages is advisable. The attempt to hasten this process by means of active or passive movements, such as riding, driving, etc., cannot be recommended. Gentle frictions in the course of the ureter, however, may give some relief in nephritic colic. For the rest, in these attacks the internal or subcutaneous use of morphia, warm baths, etc., cannot be dispensed with. If the vesicles remain impacted in the urethra, and interfere with micturition, and particularly if they cause retention of urine, we must make use of the catheter, in the fenestra of which the vesicles are readily caught. When local inflammatory manifestations arise in the vicinity of the renal echinococci, perfect rest, local extractions of blood, and applications of ice, are necessary in order to obviate suppurative processes in the cyst or its neighborhood. Operative measures will only be required for renal echinococci when their size is so great that the functions of vital organs, *e. g.*, those of the thorax, are impeded by the compression. The operations that may be employed here are the same as for echinococci of the liver, viz., simple puncture, or electro-puncture—recently so warmly recommended in England for echinococci of the liver. The few cases in which this method has so far been employed for echinococci of the kidneys were not cured. Puncture of the cyst, after adhesion of the cyst to the abdominal wall has been previously produced (Récamier), incisions after previous double puncture and leaving the canulas in situ (Simon), or puncture with injection of irritating fluids (iodine, alcohol), may also be resorted to. The cases recorded in which the operative treatment of renal echinococci was resorted to, are too few to justify a criticism of the various methods of operating. At all events, it will be advisable to begin with the mildest operation—simple puncture with the exploring trocar—as even this procedure may produce a cure. If the cyst be seated superficially enough in the lumbar region it will be most expedient to operate there, in order to avoid injury to the peritoneum.

Strongylus gigas (Rudolphi).

(Eustrongylus gigas, Palisade worm.)

LITERATURE.—*Davaine*, Traité des entozoaires. p. 267—*Leuckart*, Die menschlichen Parasiten. II. Band. Lief. 2.

This worm was known as early as the middle of the sixteenth century, and has been found in the kidneys and urinary passages, more rarely in the other organs, of carnivorous animals. It has been found in the dog, the wolf, the horse, the ox, the American marten, and in some other animals. It is exceedingly rare in man. Davaine allows only seven cases to pass as trustworthy out of sixteen collected from medical literature. Blood-clots have been several times mistaken for it. It is best to study the worm as found in animals, from which it is more easily obtained, in order to guard against mistakes.

The following description will suffice for all practical requirements. The strongylus gigas in its external appearance bears a great resemblance to a large earth-worm. The female is longer than the male; in specimens taken from the dog, respectively 36 to 64 and 31 centimetres (14 to 25 inches and 12 inches). It is distinguished from the ordinary ascaris lumbricoides by its reddish color, which evidently is produced by the bloody fluid in which it is usually found; further, by its great size and the presence of six nodules or papillæ around the mouth (ascaris lumbricoides has only three of these). In the urinary passages the parasite is found almost exclusively in the renal pelves and the calyces, very exceptionally in the ureters. The symptoms produced by the worm are the same as those caused by other foreign bodies, and are analogous especially to those caused by calculi; among them may be mentioned hæmaturia, pyuria, ischuria and strangury, and pains in the region of the kidney. Whether these parasites are discharged with the urine or not is utterly unknown, as is also the mode in which they reach the kidney. The pathologico-anatomical lesions that are recorded as produced by them are destruction of the kidney, dilatations of the renal pelvis, distention of the same with blood-clots, and pyelitis.

Pentastomum Denticulatum.

E. Wagner, Archiv. f. phys. Heilkunde. 1856. S. 581.

Only of pathologico-anatomical interest; so far observed only once in the kidney by E. Wagner, and then as a calcareous nodule, four mm. long, situated close under the renal capsule. A more detailed description is given in the article on Diseases of the Liver. In the liver the pentastomum is found more frequently.

Distoma Hæmatobium (Bilharz).

Bilharz, Zeitschrift für wissenschaftliche Zoologie. IV.—*The same*, Wiener med. Wochenschrift. 1855.—*Griesinger*, Archiv für phys. Heilkunde. 1854. S. 561. —*Leuckart*, Menschliche Parasiten. I. S. 617.

This parasite, first described by Bilharz, is exceedingly prevalent in Egypt, the Cape of Good Hope, and certainly in other hot countries also. In Egypt it is so common that Griesinger found it in 117 out of 363 bodies, but not always in equal numbers.

The female is longer than the male (respectively 18 to 19 and 12 to 14 mm. [$\frac{7}{10}$ to $\frac{15}{20}$ and $\frac{9}{20}$ to $\frac{11}{20}$ in.] in the sexually matured condition). The fore part of the body of the parasite is flattened, the posterior part cylindrical. The former bears two suckers situated not far apart. The animal probably penetrates from the intestine into the veins of the abdomen. The eggs, which are 0.12 mm. in length ($\frac{1}{285}$ in.) and 0.04 mm. ($\frac{1}{625}$ in.) in width, are pointed at one end, or provided with a pointed tooth placed on one side, but the surface is otherwise smooth.

The discharge of the eggs with the urine renders the diagnosis of distoma certain. The eggs are deposited in the urinary passages, into which they make their way through the vessels of the mucous membrane. In this way occlusions of the vessels, hemorrhage, and ulcerations of the mucosa arise. The development of the embryo begins inside the urinary passages, a long time after the deposit of the eggs. The embryos are cylindrical, with a proboscis-like, pointed anterior end, and are ciliated. Their further destinies are unknown. So long as they are found in the urine they appear to undergo no alteration and to be incapable of development. They rapidly perish in it, as well as in impure water which contains decomposed vegetable or animal matters. In pure or salt water, however, they break through their investing membranes, change their form and swim freely about. I shall not describe here the changes in the bladder which are caused by the distoma. They are very marked and severe (comp. Diseases of the Bladder). The parasites cause marked changes in the ureters also, and less frequently in the renal pelves. The ureter is constricted, and above the point of stricture is dilated. Hydronephrotic dilatation is produced. The stricture is usually formed by irregular, isolated, grayish yellow, slightly elevated patches, which are composed of a soft but very firmly adherent deposit. These have usually a sabulous feel, and consist of a quantity of dark urinary gravel and grains of uric acid, the nucleus being formed by a mass of distoma eggs. A large number of the eggs are also found free, not incrusted by the urates. They sometimes contain embryos, and are sometimes empty; now and then we also find dead animals that have escaped from the eggs.

Finally, the *Spiroptera hominis* (according to Schneider, identical with the Filaria piscium—Reichert's u. du Bois' Arch. 1862, S. 272) and the *Dactyleus aculeatus*, described by Curling (according to Schneider, probably very transparent larvæ of flies), must also be mentioned in order to warn observers against confounding them with animals accidentally present in the urine, or placed in it designedly by malingerers, and which may be mistaken for parasites of the urinary organs.

In some rare cases, moreover, in consequence of ulcerative processes, ascarides have made their way from the bowel into the pelvis of the kidney, and appear then to have been invariably mistaken for the strongylus gigas.

Anomalies in the Position, Form, and Number of the Kidneys.

The kidneys, like some other internal organs, may present certain deviations from their natural *position*, *form*, *number*, and *size*. Sometimes several of these anomalies are combined. Some of them are congenital, being *vitia primæ formationis*, while others are produced later in life, either through disease or external mechanical injuries of various kinds. Many of these variations from the normal cause no symptoms *intra vitam*, and constitute simply interesting pathologico-anatomical lesions ; others simulate various pathological conditions, and are of great diagnostic interest ; others, finally, themselves cause disturbances of health. These anomalies are often of great practical importance. They are especially dangerous when they interfere with the normal secretion and excretion of the urine. I shall consider separately the anomalies of position and the anomalies in the form and number of the kidneys.

The Abnormal Positions of the Kidneys.

The kidneys may be dislocated and fixed in their abnormal positions (*fixed dislocation of the kidneys*), or they may possess a *certain degree of mobility*. These two conditions demand a separate consideration.

The Fixed Dislocation (Dystopia) of the Kidneys.

LITERATURE.—In addition to that cited on page 543 : *Gruber*, Œsterr. Zeitschrift für prakt. Heilk. 1866.— *Weisbach*, Wiener medic. Wochenschrift, 1867.— *W. Stern*, Dissert. inaug. Berlin, 1867.—*Friedlowsky*, LX. Bd. der Sitzungsberichte der Wiener Academie, 1869.

Besides the deep position coincident with the fusion of the two kidneys, of which I will speak farther on, we also meet with a similar alteration of position in otherwise normal kidneys.

The kidney is found lying on the vertebral column, either on its own or on the opposite side, or in the median line. It is situated usually in the neighborhood of the promontory, seldom rising as high as the fourth lumbar vertebra, while, on the other hand, in a few cases it is situated deep in the pelvis, in the concavity of the sacrum. These anomalies are ascribable to a deficient energy in the movements of the embryonic rudiments of the kidney, which, up to a certain time, are situated immediately in front of the point of bifurcation of the aorta (Kupfer). At the same time the anomalous origins of the renal vessels and the neighboring vascular trunks, their usually increased number and corresponding shortness, are worthy of notice. The ureters are shortened, and run an abnormal course. The anomalies of the vessels are easily understood. When the rudimentary kidney is bound down to the spot where it originated, or its vicinity, in consequence of some pathological process, it is very probable that the vascular system gradually developing in it forms communication with the neighboring large vessels.

The form of the dislocated kidney is more or less anomalous. It is usually flattened and roundish, sometimes three- or four-cornered. The hilus is directed forward. The fœtal lobulation of the surface is usually strongly marked. The dislocation of the kidneys affects by preference the left kidney. Of 44 cases of deep position of the kidneys which Weisbach collected, in 35 the left kidney was displaced, in 8 the right, and in one both (in one case a horse-shoe kidney was deeply situated). These dislocations of the kidney seem to occur principally in men. Of 29 cases, 20 were men and 9 women.

The practical importance of this rare congenital anomaly—Stern collected altogether forty-eight cases—is in general very slight. It has never been diagnosticated during life. It would also be impossible to differentiate a congenital displacement of the kidney from a movable kidney, which later in life had become fixed in its abnormal position by pathological adhesions.

In a case observed by Hohl a deeply situated kidney presented during two parturitions a hinderance to delivery, which was finally overcome by very strong pains. The tumor could each time be felt in the vagina. However, its true nature remained unknown until death, which occurred forty years afterward, when the condition of affairs was cleared up.

When the kidneys are so deeply seated, they may easily be mistaken for other tumors in the true pelvis. An examination per rectum—if necessary, the complete rectal examination proposed by Simon—might clear up doubtful cases of this sort.

None of the other fixed displacements of the kidneys have any practical interest. I therefore pass them by, and refer the curious to the text-books of pathological anatomy.

I will here only briefly mention a case of unusually high position of the left kidney, taken from the records of the Breslau Pathological Institute (A. C. 1866. No. 32). The kidney was so situated that its upper end was higher than the spleen, and it pressed the diaphragm upward like a tumor. When looked at from in front, the highest point of the left kidney was on a level with the point of junction of the fifth rib with its cartilage; looked at from behind, the origin of the ninth rib corresponded with its upper border (Waldeyer).

The Movable (Migratory) Kidney.

History and Literature.

The clinical history of the movable kidney dates from the time of Rayer. Although earlier observers, viz., Mesué[1] and Johannes Riolan,[2] published observations on movable kidneys, which deserve credit, yet they had no influence on medical practice. After Rayer had inaugurated the clinical elaboration of this condition, his description was further enlarged by other observers, and a great amount of clinical material furnished.

The following account of the affection is based upon the following publications, in addition to the works named on page 543:

---

[1] Opera omnia. Venetiis. 1561.

[2] Manuel d'anat. et pathol. Lyon. 1562.

Fritze, Archiv. gén. 1859.—*Becquet*, ibid. 1865.—*Ferber*, Virchow's Archiv. LII. S. 95.—*Gilewski*, Oesterr. Zeitsch. f. Heilk. u. Sitzungsber. der k. k. Gesellsch. der Aertze in Wien. (Wiener medic. Presse. 1865. S. 430).—*Mosler*, Berl. klin. Wochenschrift. 1866. Nr. 41.—*Steiger*, Würz. med. Zeitschrift. VII. S. 169.—*Emil Rollet*, Path. und Therapie der beweglichen Niere. Erlangen 1866.—*Trousseau*, Mediz. Klinik. Deutsch. III. 1868. S. 554.—Inaugural dissertations of *Max Schultze* (1867) and *Th. Tzschaschel* (1872). Berlin.—*Guéneau de Mussy* Union medic. 1867. Nr. 74 u. 76, and a mass of other clinical material.

Etiology.

It is probable that movable kidneys occur far more frequently than is usually believed. Many cases of long-continued abdominal pains and obscure disturbances in the lower part of the abdomen are primarily due to movable kidneys, which will escape notice so long as an objective examination is not made. Walther of Dresden has examined a great number of persons, and found movable kidneys in many in whom the anomaly caused no symptoms whatever, so that the patients were entirely ignorant of its existence. An accurate estimate of the frequency of the anomaly is consequently impossible, since, as a rule, only those cases come to the physician's knowledge in which the anomaly causes troublesome symptoms, or in which the mobility of the kidney is accidentally discovered during an examination of the abdomen undertaken for some other reason. When, therefore, Rollet states that among 5,500 patients in Oppolzer's clinic there were 22 accurately determined cases of movable kidney, a proportion of 1 : 250, his figures must be received, as he himself admits, *cum grano salis*. According to Dietl, the movable kidney is especially frequent among the inhabitants of Poland. According to Moeller,[1] it is by no means rare in the province of Prussia, and numerous cases have been reported, particularly from the city of Braunsberg. In the Charité at Berlin, in 3,658 autopsies, movable kidneys were found five times (1 : 732).

---

[1] Berlin. klin. Wochenschrift. 1872. Nr. 37.

The statement made by Rayer that the female sex is particularly predisposed to this anomaly has been completely verified. I have collated from medical literature the reports of 96 cases, of which 82 occurred in females and only 14 in males. Most of the patients belong to the working classes, and I will soon show that this is not an accidental coincidence. In children a movable kidney is rarely met with. However, examples are not wanting. Steiner[1] found it three times: twice in girls aged six and ten years respectively, and once in a boy of nine years. The right kidney was always the one affected. It required one and one-half to three years to descend into the right lower abdominal region. In the second decade of life also floating kidneys are rarely met with; only two cases are known to me. Most of the cases happen between the ages of twenty-five and forty years, the time during which women bear children. In the great majority of the cases the right kidney is the movable one. In a certain number of cases abnormal mobility of both kidneys is found. In 91 cases that I collated from medical literature I found that the right kidney was affected 65 times, and the left 14 times, and both kidneys 12 times. Cruveilhier noticed this predilection for the right kidney. He found in women who compressed the liver by tight lacing, the right kidney sometimes in the fossa iliaca, occasionally in front of the vertebral column, and occasionally on a level with the origin of the mesentery in which it was imbedded. The kidney, when accidentally dislocated in this way, possesses a certain amount of mobility. The less frequent displacement of the left kidney is due to the fact that the left hypochondrium, which is occupied by the spleen and the fundus of the stomach, bears the pressure of the lacing with greater impunity.

The movable kidney may be either *congenital* or *acquired.* How is the latter brought about? The kidney, imbedded in its fat capsule, is situated behind the peritoneum lying upon the quadratus lumborum and the last two costal attachments of the diaphragm. In its normal condition it is held in position by the tension of the peritoneum, which passes over it, and which is attached to the anterior layer of the fat capsule, and is pretty firmly attached below and laterally to the kidney.

---

[1] Compend. der Kinderkrkh. 2 Aufl. 1873. S. 322.

When from any cause the fat disappears from the capsula adiposa, the kidney can readily be moved about in the subserous tissue. It can be demonstrated, by experiments on the cadaver, that the kidney becomes very movable when the resistance of the diaphragm is removed. Movement outwards is restricted by the resistance of the vascular trunks. It is, therefore, very natural to suppose that the predisposition to movable kidneys depends upon certain anatomical peculiarities, such as great looseness and flexibility of the peritoneum. Such a condition is produced by the wasting and partial disappearance of the fat capsule of the kidney, that is usually observed in badly nourished persons who are debilitated by chronic or severe acute disease; also by a loose attachment of the peritoneum to the posterior abdominal wall, by dragging downward of the same, due to the traction of hernias (Rayer describes a movable kidney that complicated a hernia cruralis, in which the cæcum was contained), or by relaxed and thin abdominal walls.

Moreover, the increased weight of the kidney consequent on augmentation of its volume, such as occurs in tumors of the organ, in hydronephrosis, in the formation of calculi, or in cancer, helps to make it more movable. In these cases, however, the kidney is very often fixed in its original place by inflammatory perinephritic adhesions. The kidney may also be displaced by tumors in its neighborhood, by enlargement of the liver or the spleen, especially by large leukhæmic tumors of the latter, and even by tumors of the supra-renal capsule and the pancreas. That cancerous kidneys, in consequence of their size and weight, may become movable, was maintained even by Troja, and in more recent times by Rollet (see Diagnosis).

In all these cases, the displacement of the kidney takes place slowly and gradually (so-called spontaneous dislocation).

The marked predisposition which women have to mobility of the kidney depends only to a very small extent upon the injurious action of tight lacing, the movable kidney being relatively least frequent in women belonging to the wealthier classes, by whom corsets are most constantly worn. It is excited chiefly by repeated pregnancies and deliveries. That the distention of the abdominal coverings and of the abdominal cavity, and the flabbiness of the abdominal walls, which remains after the delivery, have actually a part in the pathogenesis of the movable kidney, is evident from the fact that it is precisely women who have gone through many pregnancies who are most likely to suffer from this anomaly. Becquet believes that a hyperæmic swelling of the kidney takes place during the menstrual period, that in consequence of the monthly repetition of this condition the connective tissue surrounding the organ becomes relaxed and loosened, and that in this manner the kidney becomes free and movable. This hypothesis, however, has no real value, since the premises from which it starts are entirely unproved. On the other hand, certain injuries, heavy labor, great exertions, perhaps contusions, etc., predispose to the production of movable kidneys. It is on this account, apparently, that the working classes suffer from them so much more frequently. The dislocations induced in this manner are classed as traumatic. Oftentimes there seems to be a combination of several causes. In this respect the case reported by von Dusch[1] is very instructive. In this case the patient had given birth to eleven children, and the abdominal walls were very relaxed; the last four deliveries were so difficult that artificial aid had to be resorted to. Moreover, three years previously she had fallen down stairs, striking upon the right side, and says that she soon afterwards felt a tumor in the right hypochondrium. Finally, congenital individual predispositions to displacements of the kidney may also exist, as *e. g.*, looseness of the perinephritic connective tissue, unusual length of the renal arteries, etc.

Pathology.

Pathological Anatomy.

As the movable kidneys are never of themselves fatal, they are almost always accidental post-mortem discoveries.

---

[1] Bericht über die medicinische Poliklinik zu Heidelberg von 1857–1859.

In the movable kidneys, in which the mobility has been developed subsequently to birth, we meet with no anomalies of the vessels, they being, at the most, somewhat elongated. On the other hand, the renal capsule usually contains no fat, and the connections of the peritoneum with the renal capsule are completely loosened. The kidney can of course only be moved within a segment of a circle whose radius corresponds to the length of the renal vessels. The kidney can be displaced downward, inward, and forward. Apart from this change of site, a change in the position of the kidney usually occurs, which will vary according to the different degrees of relaxation of the peritoneum, according to the changes in the position of the body, and according to the degree of distention of the neighboring organs and the amount of pressure exercised by them. As has been already remarked, the movable kidney is generally found on the right side. In the dead body it is found in one of the different abnormal locations, from which it can usually be readily brought back to the normal position. It is generally covered by loops of intestine. If, however, the peritoneum be greatly relaxed, and the kidney sufficiently invaginated in it, it may be found immediately beneath the abdominal walls. The degree of mobility possessed by these kidneys varies. I made an autopsy in 1863 upon a woman upward of fifty years of age, who had slowly wasted away in consequence of a caries of the fourth dorsal vertebra. The body was greatly emaciated. The right kidney lay transversely across the vertebral column at its normal height, the hilus being directed backward and upward, and the convex margin downward and forward. The fat capsule was completely gone. The kidney, which was of normal configuration, could be pushed down as far as the inlet of the true pelvis. In the middle of the posterior surface of the kidney there was a cavity with smooth walls, about the size of a walnut, filled with cheesy, slimy contents; the parenchyma was otherwise pale, but normal. The renal pelvis was quite normal; the capsule easily separable. The left kidney was quite healthy. The dislocated kidney is often found imbedded in masses of old exudation. These owe their origin to previous attacks of perinephritis, the consequence of the occurrence of the so-called incarceration. Adhesive inflammations, with consequent adhesions to other organs, whereby the kidneys become fixed in abnormal locations, are seldom observed. In one case the dislocated kidney was found attached to the gall-bladder and the transverse colon by firm connective tissue.

The congenital movable kidney is distinguished from the acquired by the fact that the renal vessels are abnormal in number, origin, and course ; moreover, in individual cases, anomalies of the peritoneum or of neighboring organs are also found. Thus the kidney has been found suspended in a peritoneal fold of its own—meso-nephron—and situated near the anterior wall of the abdomen. The rectum also has occasionally been found in an abnormal position. In one of Durham's cases this condition, in a woman thirty-four years of age, permitted an abnormal mobility of the kidney. The form of the kidney is also often altered.

There can be no doubt that the movable kidneys, which are occasionally met with in abdominal ruptures, are also congenital. Thus Monro relates the history of a boy six months old in whom two tumors covered by skin existed, which could be readily pushed back into the abdominal cavity through a ring of considerable size.

Symptomatology.

The movable kidneys, in a great number of cases, cause no symptoms. In other cases the patients frequently have an indistinct feeling of uneasiness—a sensation of dragging,—as if some organ had "broken loose," and was moving hither and thither in the abdomen. These symptoms, especially when, by palpation, the patient himself discovers the "movable kidney" in his abdomen, cause great anxiety, psychical irritability, and depression of spirits. Often, however, the symptoms are more urgent ; an unpleasant sensation of pressure and weight in the belly, particularly when walking or standing, or when turning in bed, dyspepsia, nausea, which increases until emesis is produced, and colicky pains in the abdomen, set in simultaneously or come on gradually. Intense radiating pains may occur, which radiate not only toward the epigastrium, the sacral and lumbar region, and the vicinity of the navel, but also toward the intercostal spaces, into the shoulders, along the ureter, into the region of the bladder, and into the spermatic cord and the testicle, or into the labia majora. These pains are undoubtedly due to stretching of the renal nerves in consequence of the displacement of the organ. They are called forth or increased by violent active or passive movements, by walking, dancing, riding, or driving. Sometimes they are aggravated even when the patient lies on the opposite side. In addition to these pains, intense paroxysms now and then occur, to which Dietl drew particularly the attention of physicians as "evidences of incarceration." The attack is ushered in by slight chilliness or by actual chills, accompanied by great anxiety and most intense pain. The attacks may be so severe as to cause fainting and collapse. Palpation reveals a smooth and very sensitive tumor. Accurate palpation soon becomes impossible, because inflammatory manifestations have set in, with great tension, hardness, and tenderness of the abdomen. This circumscribed and more or less intense peritonitis often produces a considerable amount of exudation, which increases the extent of the dullness on percussion. In the course of a week the exudation that has been poured out is usually reabsorbed. Older observers have even reported decomposition of the kidney, and the formation of abscesses as a consequence of the incarceration. In more recent times no examples of this have been recorded. As regards the causation of these manifestations of incarceration, the most widely accepted opinion is that they are due to the irritation of the surrounding connective tissue and the peritoneum, consequent on some sudden change in the position of the kidney. Gilewski not long ago sought to explain the symptoms of incarceration on the theory of an acute hydronephrosis and pyelitis. He explains the production of the latter by assuming that when the kidney is twisted upon its own axis, the ureter is compressed, and in this way retention of urine, pyelitis, and their attendant manifestations, are produced. Gilewski saw improvement set in simultaneously with the discharge of a muco-purulent urine. We certainly cannot deny that it is possible for the manifestations of incarceration to be produced in this way; but, at all events, it does not afford a satisfactory explanation for all cases. The results of some autopsies that have been published in the meantime do not favor Gilewski's theory. It is by no means decided, as yet, whether the pyelitis is the cause or the result of the manifestations of incarceration. At all events, pyelitis is often found in the movable kidneys when the signs of incarceration have not existed, and, on the other hand, the urine, when the signs of incarceration are present, often contains no pus or any other abnormal ingredient; it presents simply the characters of a febrile urine. Hematuria has been observed in some cases. A complication with nephrolithiasis appears almost always to exist in these cases. Blood has not been found in the urine in connection with symptoms of incarceration.

The manifestations of incarceration cease as soon as the replacement of the dislocated kidney is accomplished. In addition to the above symptoms, the movable kidney often causes œdema of the lower extremities by pressure upon the vena cava ascendens. Rayer reports a case in which a movable kidney produced obliteration of the inferior vena cava. Rollet saw in one case a compression of the ascending colon produced by a floating kidney, and in such cases a disturbance in defecation may be explained by extreme compression of different portions of the intestines.

On examining the abdomen of an individual with a movable kidney, a tumor is felt, the resemblance of which to a kidney may under favorable circumstances be made out. In isolated cases (twice in Frerichs' clinic) the pulsation of the arteria renalis has been felt. Usually the outlines of the kidney cannot be completely mapped out. The tumor is found either under the free border of the ribs or lower down, near the navel, or occasionally in the region of the iliac fossa. The tumor is firm, usually somewhat sensitive, easily movable, and by proper manipulation can be forced back more or less readily into the lumbar region. The lumbar region often appears, when the kidney is displaced, somewhat flattened and sunken in. After the replacement it becomes as prominent as the other side. The percussion note over the normal site of the displaced kidney is louder, and the resistance on palpation is less than on the other side. Of course these signs also disappear when the kidney is replaced. In the great majority of the cases numerous loops of intestine, containing gas, intervene between the dislocated kidney and the abdominal wall, so that the percussion sound over the displaced kidney is tympanitic, provided no other causes of dullness on percussion, such as fæcal masses, be present. Only in very rare cases can the kidney push forward the peritoneum to such an extent as to produce a mesonephron of sufficient length to allow it, when the patient is placed in a favorable position, to come directly under the abdominal wall, where it will give a flat percussion note. In one case the kidney was felt as a tumor to the right of the navel. It caused the patient much pain. Replacement was impossible. The dislocated kidney descends somewhat during deep inspiration, and is then less easily felt, because loops of intestine are at the same time forced between it and the abdominal wall.

Diagnosis.

The diagnosis of movable kidney is based upon the facts that the palpable tumor has the form of the kidney ; that inspection, when the patient is thin, shows a sinking-in of the lumbar region corresponding to the absent kidney ; that the percussion-sound in this situation is louder and deeper than on the other side ; that the tumor can be replaced, and that then these phenomena in the lumbar region disappear. The two last signs are wanting when the dislocated kidney is bound down in its abnormal position by connective tissue adhesions. Trousseau resorted to a simple device in order to assure himself of the nature of the tumor. A peculiar kind of pain is excited by pressure upon it, and, if pressure be made on the lumbar region of the other side, a similar pain is produced. As the movable kidneys seem almost never to occur in fleshy persons, the difficulty which the accumulation of fat in and upon the belly interposes to palpation is avoided. In the majority of the cases the diagnosis seems easy, and the errors which are made are mostly due to the fact that the physician does not bear in mind the possibility of this affection. A correct diagnosis acts often as a complete remedy, since it dispels the hypochondriacal thoughts which are excited by the presence of the tumor in the abdomen. Movable kidneys are often confounded with other tumors—*e. g.*, with tumors of the gall-bladder, and with fæcal, splenic, glandular, and ovarian tumors. With regard to the last, the differentiation can only be difficult when the ovarian tumors are small and possess long pedicles. It would be too great a task to attempt to discuss here in detail the diagnostic signs of these affections, which are fully described elsewhere.[1] I have met with one case in my practice in which an echinococcus in the mesentery (there existed at the same time a large echinococcus sac in the liver) was mistaken for a floating kidney.

The diagnosis is difficult when the movable tumor is not accessible to examination by palpation. Some observers, such as Moeller, believe that the evidence furnished by palpation can rarely be obtained. He bases the diagnosis in such cases upon the characteristic nature of the lumbar neuralgia, which is distinguished by the peculiarity that active and passive movements, especially horseback exercise, increase and at times excite the pains, while they are greatly relieved when the patient assumes the dorsal position. The diagnosis in such cases is, of course, based only upon probabilities which are derived from observation of the accompanying neuralgias and the causes that excite or aggravate them. Moreover, the diagnosis meets with difficulties when the dislocated kidney has formed extensive adhesions so that it is impossible to replace it. Under such circumstances the diagnosis may be impossible if the anamnesis is also wanting. The diagnosis may also be impossible when the dislocated kidney has undergone degeneration and its original form has been lost. Such errors in diagnosis are not immaterial for practice, because they may lead to very dangerous mistakes in treatment. In one case[2] a movable kidney in a state of cancerous degeneration was mistaken for an ovarian tumor, and an operation for its extirpation was begun. In this case the renal tumor was situated directly beneath the abdominal wall in front of the intestines.

---

[1] *Vide* the appropriate chapters of this work.

[2] Lancet, 1865, March 18.

Duration, Course, Prognosis.

The affection is of long duration ; recovery does not seem to take place. Oftentimes adhesions are formed between the dislocated kidney and the neighboring organs. The movable kidney has, it seems, never been known to terminate fatally.

Treatment.

A correct diagnosis, as has been previously mentioned, is in itself of therapeutic value, because it allays the fears of the patient, who has imagined his condition to be very bad. It will also prevent not only useless, but often injurious employment of preparations of iodine and mercury to bring about resorption of the supposed tumor. For the rest the objects of the treatment must be to reduce the dislocation of the kidney and thereby relieve the symptoms produced by it, and particularly to guard against the manifestations of incarceration. The unpleasant sensations disappear at once when the organ has been successfully replaced. The manipulation by which the replacement is brought about is generally a simple one, and indeed the dislocated kidney very frequently falls back into its normal position when the patient lies quietly on her back. A light, gentle pressure, exerted by the hand upon the kidney and directed towards the lumbar region, is generally successful in replacing the gland. In order to prevent it from again leaving its place, it is necessary for the patients to wear bandages so arranged as to keep the organ in its normal position. Ordinary abdominal bandages are sometimes sufficient, but usually a bandage of strong ticking, large enough to surround the entire abdomen, is required ; this must be padded, and at the point corresponding to the tumor must contain a strong, elastic, concave pad. Guéneau de Mussy recommends a pad shaped like a square, so applied that the lower branch will keep the kidney from falling forward, and the vertical branch will keep it from slipping inward or outward. An elastic bandage similar to the elastic stockings recommended for varicose veins of the leg, may also be employed. Sometimes an apparatus resembling a truss has been employed. The most important point is to have the bandages fit well; they must be accurately fitted to each individual case. Unfortunately, they accomplish the desired end, as a rule, very incompletely, as the kidney very often slips away again, and the bandages may then cause more discomfort than the dislocated organ itself. These bandages must be worn constantly. It is evident, of course, that the patients must avoid all violent movements and all severe bodily exertions, and that any constricting articles of clothing previously worn, especially stays, must be at once laid aside. It is important to keep the bowels regular, in order to avoid constipation and the consequent injurious straining upon defecation.

If symptoms of incarceration set in, the replacement must be at once attempted. Some, like Gilewski, advise that the physician should not allow himself to be misled by the pains. Generally it is advisable to avoid violent measures. When the pains set in the patient must, in the first place, lie quietly on her back, and this alone will ease the pains considerably. The employment of a warm bath, of warm cataplasms, and of subcutaneous injections of morphia, will frequently enable the replacement to be performed with but little pain. If, however, the organ cannot be replaced, and a circumscribed peritonitis sets in, we must endeavor to cure the inflammatory affection by rest, applications of ice, opiates (internally or subcutaneously), leeches, and cupping; a renewed effort to replace the organ may then succeed, provided adhesions have not formed in the interval.

As the movable kidney frequently occurs in debilitated and emaciated individuals who are suffering from great anæmia and hydræmia, the treatment in the majority of the cases must include a supporting diet and the use of preparations of iron—in short, a tonic regimen. Some observers, among others Flemming,[1] assert that the mobility of the kidney has been cured by a tonic treatment continued for a long time.

---

[1] Brit. Med. Journ. 1869. August 21.

Anomalies in the Form and Number of the Kidneys.

LITERATURE.—In addition to the works enumerated on pages 543 and 761: *Neufville*, Arch. f. phys. Heilkunde. 1851. S. 276.—*Mosler*, Arch. f. Heilkunde. 1863, S. 289.

Apart from the anomalies in the form of the kidney consecutive to diseases of the organ, of which we have already spoken in the appropriate places, the kidneys may become flattened, compressed, and altered in form, in consequence of diseases of the neighboring organs, particularly enlargements and tumors of the same. The remaining anomalies of form are congenital, and concerning them some remarks have already been made (vid. Fixed Dislocation of the Kidney). They have just as little practical importance as the *lobulated kidney,* upon whose surface the boundaries of the several renculi are designated by shallow grooves. This is a relic of the fœtal condition, which ordinarily disappears soon after birth, but sometimes persists.

A combination of anomalous form and position of the kidney is brought about by *fusion of two kidneys* at single points, while at the same time they move nearer to one another. These partial renal adhesions may be divided into three classes. In the first the kidneys are united at their lower ends, and constitute the so-called horse-shoe kidney (Ren unguiformis, Renes arcuati, Ren soleiformis). In some cases this may be the cause of errors in diagnosis.

H. Sandwith[1] mentions a case in which it was mistaken for a dilatation of the abdominal aorta. The autopsy revealed an exostosis of the third lumbar vertebra, by which the united kidneys were pushed forward, and thus formed a visible pulsating tumor under the abdominal covering. J. B. Morgagni mentions a case where an aneurisyma aorticum was caused by the pressure of a horse-shoe kidney.

In its pure type the *horse-shoe kidney* presents only a union of the inferior apices of the kidneys, by means of more or less intervening tissue. The entire mass is situated more horizontally, with the concavity upward and the convexity downward. This form is relatively the most frequent. In the second form the union takes place in the middle by the two hili. In slight grades of the anomaly it is only a small connecting bridge which unites the two kidneys with one another. The highest grade has been observed by Meckel,[1] but even in that case the upper and lower ends were separated. The slighter grades of this anomaly are not very rare. In a few exceedingly rare cases the supernumerary renal parenchyma is not united to the two kidneys, so that the middle piece constitutes an independent kidney, which receives blood from both the lateral parts, but also possesses in part independent vessels. The third form, in which the two kidneys grow together at their upper ends, is the rarest of the three forms. Meckel states, in describing the horse-shoe kidney, that in rare cases the convexity is directed upward and the concavity downward. This is the lowest grade of the third form. In the higher grades the amalgamation may extend from the upper ends until the bodies of the two kidneys coalesce and only the lower ends are separate. Sometimes these too are only separated by a shallow depression. I append an account of one of these exceedingly rare cases, observed by Neufville, on account of the remarkable clinical manifestations which it caused. Although the diagnosis could not be made out during life, still this case affords fresh proof of the value of the old rule, that it is necessary to be very cautious when we have to deal with diseases whose nature is not quite clear.

---

[1] Schmidt's Jahrbb. XLIV. S. 186.

The patient was a woman aged twenty-five years. Until she reached that age the horse-shoe kidney, in spite of its position transversely across the vertebral column, the aorta, and the inferior vena cava, had, as a rule, caused no disturbances, because during the pulsations of the abdominal aorta the isthmus was raised, and the pressure of the kidney on the vena cava was thus moderated and rendered harmless. Suddenly, however, in consequence of congestion of the amalgamated kidneys, a compression was exerted upon the vessels, which caused thrombosis of the large veins, and death resulted from complete arrest of the circulation.

Under certain circumstances, in consequence of the anomalous form and position of the kidneys, the escape of the urine may be prevented and a fatal termination result.

---

[1] Path. Anat. I. S. 616.

W. Koster[1] relates the history of a woman who was brought to bed in the sixth month of her second pregnancy, and who died from uncontrollable vomiting. At the autopsy a greatly enlarged horse-shoe kidney was discovered lying upon the vertebral column. It possessed two separate renal pelves and two ureters, of which one was plugged with stinking pus, and the other with thick mucus. During the pregnancy the ureters had been compressed by the enlarged uterus, and thus retention of urine in the renal pelvis was caused. During the first pregnancy the kidneys were still healthy, and the effects of retention of urine had not yet developed. In the second pregnancy a pyelonephritis rapidly caused death.

Moreover, B. v. Langenbeck states that he has in a few cases seen children die suddenly with brain symptoms, probably uræmic, in whom the necropsy revealed the presence of horse-shoe kidneys.

Kidneys presenting these anomalous forms are usually also found in abnormal sites. Sometimes they have descended into the true pelvis, and lie in the cavity of the sacrum ; sometimes they are situated a little to one side, and present irregular, nodular masses, in which now and then inflammation and suppuration occur. In such cases, as Cruveilhier demonstrated, the renal abscess may perforate into the rectum. With regard to the origin of these fusions of the kidneys, they were thought, from the time of Meckel until quite recently, to be due to an arrest of development, as it was universally believed that in the fœtal state the two kidneys were fused together. This view had to be abandoned when Kupfer proved that the kidneys are formed in pairs. Up to a certain time they are located immediately in front of the point of bifurcation of the aorta, and are in contact with one another in the median line. If at this period they coalesce, a fusion of the organs is the result. This growing together is all the more probable, since it is similar to the process by which the separate lobes, of which the embryonic kidney consists, gradually unite with each other and form a body with a smooth surface, the fusion of the renal lobes always commencing in the cortical substance.

Besides the coalescence of the two kidneys into one renal body, we also meet with cases in which one kidney is entirely absent. These cases must not be confounded with the cases of great congenital atrophy of one kidney, dependent, for instance, on rudimentary development of the renal vessels, which are sometimes observed.

---

[1] *Virchow-Hirsch*, Jahresb. 1867.

In the winter of 1874 I met with an instance of this in the body of a man twenty-four years of age, who had succumbed to a general paralysis. At the upper end of the right ureter, which opened into the bladder in the normal position, a pale reddish mass of connective tissue, surrounded by considerable adipose tissue, was found. This mass was smaller than the normal supra-renal gland. No glomeruli and no uriniferous tubules could be discovered in it. On the other hand, the ureter and the renal pelvis were connected with the mass, although they, as well as the renal artery, were exceedingly narrow. The left kidney was considerably enlarged.

When the kidney is really and entirely absent, the corresponding ureter will also invariably be absent. There may be an apparent absence of the kidney in those cases where the ureter opens normally into the bladder, but the corresponding kidney has been forced over to the opposite side, and has fused with the organ of that side.[1] These anomalies may be explained by excessive mobility of the embryonic rudiments of the kidney. It is a rare thing for the ureter of a solitary kidney to cross over to the opposite side before it opens into the bladder.[2] Persons with a single kidney frequently present absolutely no disturbances of the renal functions or of the excretion of urine. The existing kidney is nearly always hypertrophied, and, so long as it remains healthy, the secretion of the urine proceeds without demonstrable disturbances. If, however, this single kidney becomes inflamed, or its ureter be obstructed by an impacted calculus or compressed by a tumor, the most alarming disturbances supervene. Of 29 cases of solitary kidney which Roberts collected, 22 occurred in males and 6 in females; in one case the sex was not stated. The age was accurately given in 18 cases: one boy was 7 days old; another was 7 years of age; two of the cases were 15 years of age; four were between 20 and 30; three between 30 and 40; four between 40 and 50; two were 60, and one 65. Of the remainder, it is only stated that they were adults. In 16 cases the left kidney was absent, in 12 the right. In 19 cases the defect was congenital; in 3 cases it was produced by destruction of the kidney in consequence of disease. In the remaining cases no information is given on this point. The renal vessels, like the ureter, are almost invariably absent on the affected side when the defect is congenital. Meschede[1] describes a case where a remnant of the corresponding ureter was present. Similar observations have been recorded by Paulicki, Muenchmeyer, and others. Occasionally, when there is but one kidney, the number of the arteries and ureters is increased. In twenty-four cases the causes of death were specified; in twelve of these cases the death was essentially due to the anomaly, being directly due in ten to the development of a renal calculus and plugging of the ureter of the single kidney, and in two to the pressure of a cancerous tumor upon the kidney.

---

[1] Sandifort, *J. F. Meckel*, l. c. p. 625.

[2] *Foerster*, Virch. Archiv. XIII. S. 375.

Hachenberg[2] has recently reported the case of a soldier, twenty-six years of age, in whom the right kidney was completely absent. Inflammation and suppuration set in in the greatly enlarged left kidney, and during the last five days of life complete anuria existed, in consequence of which uræmic manifestations were developed.

Diseases of the Renal Vessels.

LITERATURE.—The works enumerated on page 543, and *Virchow*, Gesammelte Abhandlungen. S. 470, 556.—*Beckmann*, Würzburger Verhandlungen. IX. S. 201.—*Rosenstein*, Virchow's Archiv. XII.—*O. Pollack*, Wiener med. Presse. 1871. Nr. 18.—*Ollivier*, Arch. de physiologie. 1873. p. 43.

Diseases of the Renal Artery.

Pathology.

As the principal diseases of the arteria renalis and its branches—amyloid degeneration, occlusion with consecutive necrosis, infarction or abscess, congenital and acquired constrictions—have already been described in detail, we have only to add a few remarks on them here.

---

[1] Virchow's Archiv. XXXIII. S. 547.

[2] Berl. klin. Wochenschrift. 1872. Nr. 22.

In the first place, the *atheromatous degeneration* of the renal artery is, as a rule, only a local manifestation of a universal sclerosis of the arteries; not unfrequently, however, the arteria renalis is not involved in the general sclerosis. It is, nevertheless, far more frequently affected than many authors believe. Ollivier, in particular, makes a great mistake when he claims that he was the first to call attention to sclerosis of the arteria renalis. The arterial sclerosis may extend to the smaller arterial twigs, and cause disturbances of nutrition in the renal parenchyma, in consequence of the diminished supply of blood.

Aneurisms of the renal artery are very rare. In the few cases that have been accurately observed the chief symptoms were severe pains in the lumbar region, pulsating tumor, and hæmaturia (not constant). Rupture of the sac usually occurs. In Ollivier's case, at the autopsy of a man seventy-two years of age, an aneurism as large as a filbert was found at the point of bifurcation of the arteria renalis dextra; the small arteries down to a certain size were atheromatous, and presented aneurismal dilatations. In consequence of the bursting of such small aneurisms, numerous hemorrhages into the renal pelvis had occurred during the six years that the affection had lasted. The blood was discharged per urethram, the passage being sometimes painless and sometimes attended by severe pains. The clots remained in the renal pelvis and excited a nephropyelitis (comp. above, p. 565), which led to destruction of the greater part of the right kidney. The patient died of a pneumonia. According to Ollivier, processes of this sort often give rise to hæmaturia in old persons.

Diseases of the Vena Renalis.

Thrombosis of the vena renalis is the affection which particularly comes under consideration here. The thrombosis may extend from the vena cava or spermatica into the vena renalis, but it is more frequently secondary to diseases of the renal parenchyma. In the latter case it extends usually only to the hilus, seldom as far as the vena cava.

Of the different forms of thrombosis that are met with in the vena renalis, the most frequent are the *marantic* and those due to *compression.*

Of the various affections of the kidney, in which occlusion of the renal veins is met with, the most common is amyloid degeneration; next in order of frequency comes diffuse nephritis, then extensive neoplasms of the kidney, particularly *carcinoma*, and also tumors in the neighborhood (glandular masses in the hilus renalis, retroperitoneal tumors, etc.), and, finally, enlargements of the more deeply situated organs (pregnant uterus, ovarian tumors, etc.).

In all the above-mentioned affections the thrombosis is due to *compression.* The compressing agent is to be found either in the swelling of the renal parenchyma, or in the tumors of the organ itself or of the surrounding parts.

The *marantic* thrombosis of the renal veins is met with in general cachectic conditions, during which, as experience teaches, thromboses are frequently developed. This cause, moreover, has a not unimportant influence in the pathogenesis of a number of the thromboses that are due to compression. Beckmann has particularly called attention to the cases of thrombosis of the renal veins that occur in infants who have become marantic through cholera infantum. Occlusions of the renal veins are also observed in other cachectic conditions, *e. g.*, in carcinoma ventriculi. An extension of the thrombosis from the vena cava or spermatica is met with particularly in cases of puerperal fever in connection with uterine and crural phlebitis (Cruveilhier, Anat. pathol. Livr. 36).

Finally, we must mention the thrombosis of the renal veins, which is developed as a result of *traumatic nephritis and in consequence usually of very severe injuries of the kidney.* Here the compression exerted by the swollen parenchyma is probably the essential agent in the production of the thrombosis of the veins. Cases of this sort have been repeatedly described, for instance, by Poland and Moxon quite recently.[1] In their cases thrombi were found at the same time in the arteries; the appearance of arterial thrombi after injuries of the renal artery has already been spoken of on page 608.

---

[1] Guy's Hospital Rep. [3] XIV. p. 85–96 and 99–111. Refer. in Centralbl. der med. Wissensch. 1869. S. 489 and 504.

The occlusion, as in thromboses of other parts, is sometimes complete, sometimes incomplete, the thrombus in the latter case being attached to one side of the wall, or hollowed out in the centre so as to form a canal; the thrombi may be recent and dark red in color, or old and pale, crumbling and softened, or tough, firm, and organized. The left vein is more frequently affected. If the trunk alone is involved, a collateral circulation is usually developed, but that is impossible when at the same time all the small ramifications of the vein are occluded.

Clinically the history of thrombosis of the renal veins has been very little studied. Pollack diagnosticates thrombosis of the renal veins in infants when after an attack of diarrhœa they become jaundiced, when a marked diminution in the quantity of urine secreted sets in, and when finally, blood, albumen, and casts, are found in the urine. Out of twelve cases Pollack saw two cases recover.

According to the observation of Leudet, however, even when the renal veins are occluded on both sides, a compensating collateral circulation may possibly be established through dilatation of the veins of the renal capsules and the ureters.

When this compensation occurs, the mere thrombosis of the renal veins will cause no alterations in the urine and no general disturbances. If, however, a collateral circulation is not established, we must expect *à priori* the most marked disturbances of the renal functions, especially since Oppolzer taught that hæmaturia and albuminuria are the invariable consequences of thrombosis of the vena renalis, and that later the secretion of the diseased kidney entirely ceases; the cases of Pollack may be ascribed to those forms of thrombosis of the vena renalis where no collateral circulation was established. However, the symptoms on which he relies for the diagnosis intra vitam appear to me to be insufficient, because they might also be produced in other ways.

END OF VOLUME XV.

INDEX.

Abele, 667.

Abortions in nephrolithiasis, 711.

Abscess in movable kidney, 770; in cellular tissue, in chronic parenchymatous nephritis, 348; in etiology of perinephritis, 588; of bladder, in etiology of hydronephrosis, 647.

Abscess, Renal. See under Inflammation of the Kidneys.

Acid, benzoic, in acute parenchymatous nephritis, 302; hydrochloric, in acute parenchymatous nephritis, 301.

Age in etiology of amyloid degeneration of kidneys, 504, 616; of cancer of the kidney, 661; of chronic parenchymatous nephritis, 335; of movable kidney, 765; of nephrolithiasis, 691; of nephrophthisis, 578; of perinephritis, 589; of pyelitis, 565; of renal cirrhosis, 410; of renal cysts, 640.

Air, influence of atmospheric, in chronic ulcerations in etiology of amyloid degeneration of kidneys, 500.

Albuminuria as a symptom of disease, 30; in acute hyperæmia of kidneys, 193; in acute parenchymatous nephritis, 276; in acute parenchymatous nephritis of pregnancy, 316; in amyloid degeneration of kidneys, 508, 625; in cancer of kidney, 675; in chronic parenchymatous nephritis, 359; in fatty degeneration of kidneys, 604; in ischæmia, 234; in nephrophthisis, 581; in pyelitis, 571; in renal cirrhosis, 435, 470; in renal cysts, 640.

Alcohol in etiology of renal cirrhosis, 412.

Alkalies and alkaline salts in nephrolithiasis, 733.

Alum in hæmaturia following cancer of kidney, 684; in pyelitis, 576.

Ammon, 578, 584.

Amyloid degenerations in nephrolithiasis, 722.

Amyloid Degeneration of the Kidneys, 496; history, 496; etiology, 498; course of the disease, 504; duration, 516; pathological anatomy, 517; analysis of the symptoms, 523; prognosis, 529; diagnosis, 533; treatment, 536; *complications of amyloid degeneration of the kidney with chronic parenchymatous nephritis and with contracting kidney*, 538.

Amyloid degeneration of the kidney. See also under Degenerative Processes of the Kidneys.

Anæmia in renal cirrhosis, 422; progressive pernicious, in etiology of fatty degeneration of kidneys, 603.

Anæsthetics in acute parenchymatous nephritis, 303.

Angioma of kidney. See under Tumors of the Kidneys.

Animal Parasites of the Kidneys, 742.

The Echinococci of the Kidneys, 742; bibliography and history, 742; etiology, 743; pathological anatomy, 745; symptomatology, 748; diagnosis, 752; duration, course, prognosis, 755; treatment, 756.

Strongylus Gigas (Rudolphi), 757; bibliography and general description, 757.

Pentastomum Denticulatum, 758.

Distoma Hæmatobium (Bilharz), 758; bibliography and general description, 758.

Spiroptera Hominis and Dactyleus Aculeatus, 760.

Anomalies in the Position, Form, and Number of the Kidneys, 761.

The Fixed Dislocation (Dystopia) of the Kidneys, 761; bibliography, 761; general description, 762.

The Movable (Migratory) Kidney, 763; bibliography and history, 763; etiology, 764; pathological anatomy, 767; symptomatology, 769; diagnosis, 772; duration, course, prognosis, 774; treatment, 774.

Anomalies in the Form and Number of the Kidneys, 776; bibliography, 776; *lobulated and horse-shoe kidney*, 776; *absence of the kidney*, 778.

Anthrax in etiology of acute parenchymatous nephritis, 253.

Anuria in cancer of kidney, 674; in hydronephrosis, 651; in nephrolithiasis, 711.

Apoplexy, cerebral, in renal cirrhosis, 417, 455.

Arthritis in nephrolithiasis, 720.

Asthma in uræmia, 111.

Atrophy, Granular, of the Kidney. See Renal Cirrhosis.

BABBE, J. M., 252, 272.
Bacteria in pyelitis, 568; in suppurative nephritis, 551.
Baillie, 658, 742, 743.
Ballard, 673.
Bamberger, 176.
Barker, 747.
Barreswill, 139, 153.
Bartels, 608.
Bartels on Structural Diseases of the Kidney, and General Symptoms of Renal Affections, 3.
Barthez, 692, 721.
Basham, 196.
Baths in amyloid degeneration of kidneys, 537; in chronic parenchymatous nephritis, 404; in nephrolithiasis, 732; in nephrophthisis, 586; in renal cirrhosis, 490; hot, in acute parenchymatous nephritis, 298; hot, in ischæmia, 244; various kinds of, in dropsy following passive hyperæmia of kidneys due to heart-disease, 219; warm, in acute hyperæmia of kidneys, 196.
Bayer, Ottomar, 84, 86, 349.
Beale, 701.
Beckmann, 549, 570, 618, 782.
Bequet, 767.
Beer, 180, 351, 456, 621, 622.
Beer in nephrolithiasis, 731.
Belina-Swioutkowsky, 496.
van Beneden, 743.
Beneke, 734.
Bennet, 586.
Bernard, Claude, 38, 55, 139, 153.
Bernhardt, 618.
Berzelius, 38, 687.
Biermer, 104, 291, 603.
Bilharz, 743, 758.
Bilin, waters of, in nephrolithiasis, 734.
Bilirubin formations in nephrolithiasis, 695.
Billroth, 146, 551.
Binz, 734.
Blackall, 35, 334.
Bladder, affections of, in etiology of hydronephrosis, 647; of pyelitis, 564.
Blau, 614.
Blood, changes in, in renal cirrhosis, 449.
Blood-coagula in etiology of pyelitis, 565.
Blood-letting in acute parenchymatous nephritis, 302; in nephrolithiasis, 734; in renal cirrhosis, 492, 495.
Blood, transfusion of, in renal cirrhosis, 496.
Blood-vessels, intestinal, changes in, in amyloid degeneration of kidneys, 522.
Bock, 222.
Bockendahl, 308, 312.
Boeck, 408.
Boedeker, 747.
Boerhave, 693, 720.
Boettger, 685.
Bones, affections of, in cancer of kidney, 677; in chronic parenchymatous nephritis, 348; in renal cirrhosis, 455; in etiology of amyloid degeneration of kidneys, 499, 612.
Bostock, 97, 167.
Bourneville, 146.
Bowman, 377.
Braidwood, 665.
Braun, 311, 316, 320.
Bremser, 743.
Briançon, 749.
Bright, Richard, 35, 111, 122, 163, 167, 169, 172, 185, 202, 245, 266, 401, 462, 659, 666, 673, 678, 679, 719.
Brinton, W., 660, 674, 684.
Bristowe, 678.
Brodie, 704, 711, 714.
Brown-Séquard, 38.
Budd, 610, 669.
Buhl, 54, 127, 227, 228, 232, 233, 235, 238, 239, 241, 243, 257, 593.
Burkart, A., 75.
Burns in etiology of acute parenchymatous nephritis, 256; of fatty degeneration of kidneys, 608.

CALCULI in other organs in nephrolithiasis, 722.
Calculus, Renal. See under Foreign Bodies in the Kidney.
Camphor in acute hyperæmia of kidneys, 196; in ischæmia, 244; in passive hyperæmia of kidneys due to heart-disease, 217.
Cancer of the Kidney. See under Tumors of the Kidney.
Cancer in etiology of pyelitis, 563; of amyloid degeneration of kidneys, 614.
Canstatt, 38, 40.
Cantharides in etiology of acute hyperæmia of kidneys, 191; of acute parenchymatous nephritis, 247; of pyelitis, 563; of suppurative nephritis, 546.
Carbolic acid in pyelitis, 576.
Carbonate of lime, formations of, in nephrolithiasis, 697.
Carbonic oxide, poisoning by, in etiology of fatty degeneration of kidneys, 603.
Carbuncle in etiology of pyelitis, 563.
Cardamon, oil of, application of, in etiology of acute hyperæmia of kidneys, 191.
Carlsbad, waters of, in nephrolithiasis, 735.
Caseous Inflammations of the Kidney, the Renal Pelves, and Ureters. See under Inflammations of the Kidney.
Casts, renal, description of, 75; in acute parenchymatous nephritis, 279; in acute parenchymatous nephritis of pregnancy, 316; in amyloid degen-

eration of kidneys, 511, 625; in cancer of kidney, 674; in chronic parenchymatous nephritis, 356, 379; in fatty degeneration of kidneys, 604; in ischæmia, 236; in nephrophthisis, 581; in renal cirrhosis, 448.

Catching cold in etiology of acute parenchymatous nephritis, 251; of chronic parenchymatous nephritis, 326; of nephrophthisis, 578; of perinephritis, 588; of pyelitis, 565; of suppurative nephritis, 546.

Cathartics in acute hyperæmia of kidneys, 196; in acute parenchymatous nephritis, 300; in nephrolithiasis, 734; in dropsy following passive hyperæmia of kidneys due to heart disease, 218.

Celsus, 544, 552.

Chills in nephrophthisis, 584; in perinephritis, 594; in pyelitis, 573; in suppurative nephritis, 552.

Chloral hydrate in acute parenchymatous nephritis, 303; in renal cirrhosis, 496.

Choleraic affections in etiology of ischæmia of kidneys, 222; of pyelitis, 563.

Chomel, 659.

Christison, 85, 97, 111, 154, 164, 167, 245.

Chyluria, 82.

Cirrhosis, Renal. See Renal Cirrhosis.

Civiale, 688, 692, 732, 733.

de Claubry, Gaultier, 705.

Cloudy Swelling of Kidneys See under Degenerative Processes of the Kidneys.

Cod-liver oil in amyloid degeneration of kidneys, 633; in nephrophthisis, 586.

Cohnheim, 46, 62, 64, 133, 223, 224, 226, 230, 231, 232, 262, 280, 282, 498, 544, 549, 550, 605, 606, 608.

Colberg, 64, 95, 346, 373.

Cold in hæmaturia following cancer of kidney, 684; in perinephritis, 601; in suppurative nephritis, 561.

Colocynth in acute parenchymatous nephritis, 300.

Concretions in ureter or pelvis of kidney in etiology of hydronephrosis, 645.

Constipation in perinephritis, 594.

Contracting Kidney, Genuine. See Renal Cirrhosis.

Convulsions in acute parenchymatous nephritis of pregnancy, 318; in amyloid degeneration of kidneys, 518; in chronic parenchymatous nephritis, 342; in hydronephrosis, 652; in nephrolithiasis, 710; in renal cirrhosis, 415, 426; in uræmia, 109.

Copaiba in nephrolithiasis, 732, in etiology of pyelitis, 563.

Cornil, 638, 666, 677.

Cotugno, 34, 163.

Creasote in vomiting of renal cirrhosis, 494.

Crocq, 110.

Crosse, 700.

Cruikshank, 34.

Cruveilhier, 658, 663, 765, 778, 782.

Cubebs in nephrolithiasis, 732; in etiology of pyelitis, 563.

Curling, 760.

Cystic Degeneration and Cysts of Kidney. See under Tumors of the Kidneys.

Cystic oxide formations in nephrolithiasis, 701.

DACTYLEUS ACULEATUS. See under Animal Parasites of the Kidneys.

Davaine, 743, 744, 747, 757.

Dawson, 739.

Degenerative Processes of the Kidneys, 602.

CLOUDY SWELLING AND FATTY DEGENERATION, 602; bibliography, 602; etiology, 602; pathological anatomy, 603 symptomatology, 604; *hemorrhagic infarction of the kidneys*, 604, etiology, 605; pathological anatomy, 606; symptomatology, 606; course. progress, and treatment, 607.

NECROSIS OF THE KIDNEY, 608; etiology, 608; pathological anatomy, 608.

AMYLOID DEGENERATION OF THE KIDNEY, LARDACEOUS OR WAXY KIDNEY, 609; bibliography, 609; history, 610; etiology, 612; pathological anatomy, 616; symptomatology, 624; complications and sequelæ, 629; diagnosis, 630; duration, termination, and prognosis, 631; treatment, 632.

Delivery in acute parenchymatous nephritis of pregnancy, 324.

Diabetes mellitus in etiology of pyelitis, 563.

Diagnosis. See different diseases.

Diaphoresis in acute parenchymatous nephritis, 297; in chronic parenchymatous nephritis, 401; in dropsy following passive hyperæmia of kidneys due to heart-disease, 219; in nephrolithiasis, 732.

Diarrhœa in renal diseases, 154; in amyloid degeneration of kidneys, 513; in cancer of kidney, 676; in nephrophthisis, 584; in pyelitis, 572; in renal cirrhosis, 425.

Dickinson, 183, 336, 411, 412, 414, 457, 504, 537, 575, 576, 615, 623, 624.

Diet in acute parenchymatous nephritis, 296; in amyloid degeneration of kidneys, 587; in chronic parenchymatous nephritis, 406; in nephrolithiasis, 730; in nephrophthisis, 586; in renal cirrhosis, 490; in suppurative nephritis, 561.

Dietl, 764, 770.

Diffuse Diseases of the Kidneys, 163; history. 163; classification, 189; for various diseases, see Hyperæmia, Ischæmia, etc.

Digestion, disturbances of function of, in renal diseases, 151; in amyloid degeneration of kidneys, 628; in cancer of kidney, 676; in chronic parenchymatous nephritis, 341; in movable kidney, 769; in nephrophthisis, 584; in perinephritis, 594; in renal cirrhosis, 422; in suppurative nephritis, 532; in uræmia, 110.

Digitalis in acute parenchymatous nephritis, 301; in passive hyperæmia of kidneys due to heart-disease, 217; in renal cirrhosis, 491.

Diphtheria in etiology of acute parenchymatous nephritis, 251; of pyelitis, 563.

Distoma hæmatobium of the kidneys. See under Animal Parasites of the Kidneys.

Distoma hæmatobium, eggs of, in etiology of nephrolithiasis, 691.

Dittell, 712.

Diuretics in acute parenchymatous nephritis, 300; in chronic parenchymatous nephritis, 406; in nephrolithiasis, 732; in passive hyperæmia of kidneys due to heart-disease, 217; in etiology of pyelitis, 563; of suppurative nephritis, 546.

Doederlein, 668, 670, 675, 683.

Dropsy accompanying renal diseases, 96; in acute parenchymatous nephritis, 284; in acute parenchymatous nephritis of pregnancy, 312; in amyloid degeneration of kidneys, 511, 627; in chronic parenchymatous nephritis, 336, 365; in renal cirrhosis, 422, 455, 470.

Duffin, 587.

Durham, 739, 769.

von Dusch, 767.

Dysentery in etiology of acute parenchymatous nephritis, 254.

EBERTH, 636.

Ebstein on the Affections of the Renal Pelves and the Ureters, 543.

Echinococci of the Kidneys. See under Animal Parasites of the Kidneys.

Edlefsen, 20, 27, 30, 39, 106, 370, 510, 513.

Elliottson, 168.

Emaciation in amyloid degeneration of kidneys, 624; in renal cirrhosis, 422; in etiology of movable kidney, 766.

Embolisms in etiology of fatty degeneration of kidneys, 603; of suppurative nephritis, 546.

Ergot in hæmaturia following cancer of kidney, 684.

Erichsen, 640.

Erysipelas in etiology of acute parenchymatous nephritis, 253.

Esmarch, 655.

d'Espine, Marc, 660, 662.

Etiology. See different diseases.

Eustrongylus Gigas of the Kidneys. See under Animal Parasites of the Kidneys.

Eye, affections of, in acute parenchymatous nephritis of pregnancy, 320; in renal cirrhosis, 421, 456, 476; in uræmia, 110.

FALK, 256.

Faludi, 667, 672.

Fatty Degeneration. See under Degenerative Processes of the Kidneys.

Fehr, 616.

Ferini, 278.

Fever in acute parenchymatous nephritis, 273; in cancer of kidney, 677; in nephrolithiasis, 710; in nephrophthisis, 584; in perinephritis, 594; in pyelitis, 571; in suppurative nephritis, 552; intermittent, in etiology of amyloid degeneration of kidneys, 614.

Fibrin formations in nephrolithiasis, 708.

Fibroma of Kidney. See under Tumors of the Kidneys.

Fischer, 55, 245, 253.

Fischer, H., 613.

Fistulæ following nephrolithiasis, 718.

Fleisch, 128.

Flemming, 669, 671, 775.

Foreign Bodies in the Kidney, the Pelvis of the Kidney, and the Ureter, 687.

NEPHROLITHIASIS, 687; history and bibliography, 687; etiology, 689; pathological anatomy, 694; deposits and concretions in the kidneys, the renal pelves, and the ureters, 694; pathological changes produced in the kidney, the pelvis of the kidney, and the ureter, by renal concretions, 705; symptomatology, 708; complications and sequelа, 720; diagnosis, 724; duration, terminations, and prognosis, 728; treatment, 730.

Foreign bodies in etiology of hydronephrosis, 645; of pyelitis, 563; of suppurative nephritis, 545.

Form, Abnormal, of the Kidneys. See under Anomalies in the Position.

Fourcroy, 687.

Fraenkel, 651, 657.

Frank J. P., 692, 713.

Frank, Peter, 720, 722.

Franqué, O. V., 29.

Fremitus, hydatid, in echinococci of the kidney, 749.

Frerichs, 112, 113, 126, 130, 174, 177, 182, 232, 305, 309, 324, 352, 392, 399, 463, 573, 661, 696, 744, 749, 751, 771.

Friedreich, 92, 611, 617, 623, 685.

Friedrichshall bitter water in nephrolithiasis, 736.

Froriep, 697.

GAIRDNER, 673.

Gamboge in acute parenchymatous nephritis, 300.

Garrod, 705, 720, 721, 734.

General Symptoms of Renal Disorders, 3; *local symptoms*, 4; pain, 4; inspection, 5; palpation, 6; percussion, 8; *functional symptoms, which are proffered by the quantity and quality of the urine*, 10; quantity, 12; color and other general features, 17; specific gravity, 19;

chemical composition in diseases of the kidney, 23; reaction, 23; urea, 26; albuminuria, 30; chyluria, 32; doctrine of albuminuria, 34; hæmaturia, 66; the formed elements of the urine in renal disease, 72; urinary casts, 75; *disorders manifested by the blood, by the general nutrition, and especially by the nervous system, when the kidneys cease to perform their functions properly*, 96; dropsy that accompanies renal diseases, 96; its distribution, 107; uræmia, 109; convulsions, 109; amaurosis, 110; mental and digestive disturbances, 110; respiratory disturbances, 111; the causes, 112; symptoms due to disturbances of the general nutrition produced by renal disease, 150; dyspepsia, 151; vomiting, 152; diarrhœa, 154; hemorrhages, 156.
Gerhardt, 10, 39, 40, 52, 632.
Gilewski, 770, 771, 775.
Gintrac, 558, 668.
Gjoer, 680.
Glioma of Kidney. See under Tumors of the Kidneys.
Gluge, 666.
Goetze, 742.
Goldbaum, 235.
Gonorrhœa in etiology of renal cirrhosis, 414.
Gorup-Besanez, 21, 83.
Gosselin, 146, 157.
Gout in etiology of renal cirrhosis, 414.
Granulation Tissue Formations in Kidney. See under Tumors of the Kidneys.
Grape-cure in nephrolithiasis, 736.
Gravel, Renal. See under Foreign Bodies in the Kidney.
Graves, 86, 169, 171.
Gravity, specific, of urine, 19; in amyloid degeneration of kidneys, 507, 625; in chronic parenchymatous nephritis, 357; in ischæmia, 236; in perinephritis, 597; in pyelitis, 571; in renal cirrhosis, 434.
Greenhalgh, 681.
Gregory, James, 35, 168.
Griesinger, 222, 239, 691, 758.
Gubler, 170.
Gull, W., 177, 184, 409, 410, 457, 459, 460, 461, 463.
Gummy Tumors of Kidney. See under Tumors of the Kidneys.
Gunn, 739.
Gusserow, 603.

Hachenberg, 780.
Hæmatoidin formations in nephrolithiasis, 695.
Hæmaturia as a symptom of kidney disease, 66; in acute hyperæmia of kidneys, 193; in acute parenchymatous nephritis, 278; in amyloid degeneration of kidneys, 510; in cancer of kidneys, 670; in chronic parenchymatous nephritis, 357; in echinococci of the kidneys, 751; in ischæmia, 237; in movable kidney, 771; in nephrophthisis, 581; in pyelitis, 571; in renal cysts, 640; in suppurative nephritis, 553.
Hæmorrhoidal flux, suppressed, in etiology of suppurative nephritis, 546.
Hahn, 636.
Hallé, 588.
Hamernik, 232, 241.
Hamilton, 245.
Hammond, 38.
Headache in pyelitis, 572; in renal cirrhosis, 420, 476.
Heart, changes in, in amyloid degeneration of kidneys, 514, 523, 628; in chronic parenchymatous nephritis, 367; in renal cirrhosis, 418, 457, 454, 462; diseases of, in etiology of passive hyperæmia of kidneys, 199.
Hebra, 405.
Heer, 558.
Heidenhain, 14, 119, 213, 377, 469.
Heinemann, 693.
Heinrich, 730.
Heintz, 747.
Heller, 9, 76, 85, 252, 272, 306, 322, 380, 520, 675, 691, 699, 700, 703, 733, 737.
van Helmont, 96.
Hemorrhages in amyloid degeneration of kidneys, 629; in renal cirrhosis, 430; in etiology of pyelitis, 565.
Hemorrhagic diathesis in renal diseases, 156.
Henle, 75, 82, 87, 113.
Henoch, 277, 278.
Heredity in etiology of nephrolithiasis, 692.
Herniæ in etiology of movable kidney, 766.
Herrmann, Max, 50, 61, 259.
Hertz, 640.
Hervieux, 146.
Heschl, 637.
Heusinger, 642.
Hildebrand, 645, 652, 654.
Hillier, 657.
Hippocrates, 96, 709.
Hirsch, August, 691.
Hirschsprung, 661.
Hirtz, 671.
History. See different diseases.
Hodann, 695, 703.
Hoffmann, Carl, 622, 685.
Hohl, 763.
Hoogeweg, 696.
Hoppe-Seyler, 116, 127.
Hotz, 655.
Huber, 583.
Hufeland, 735.
Huppert, Max, 48, 319, 448.
Hydronephrosis. See under Tumors of the Kidneys.

Hyperæmia of the Kidneys, 190.
  Active Hyperæmia, 190; etiology, 191; anatomical changes, 192; symptomatology, 192; diagnosis, 194; treatment, 196.
  Passive Hyperæmia, 197; etiology, 199; heart-diseases, 199; lung-diseases, 200; pathological anatomy, 201; symptomatology, 204; course and terminations, 213; diagnosis, 214; prognosis and treatment, 216.

Immermann, 603.
Infarction, Hemorrhagic, of Kidneys. See under Degenerative Processes of the Kidneys.
Inflammations of the Kidney, of the Pelvis of the Kidney, and of the Perinephritic Tissues, with Termination in Suppuration, 543.
  Suppurative Nephritis, Renal Abscess, 543; bibliography, 543; introductory remarks and history, 544; etiology, 545; pathological anatomy, 547; symptomatology, 552; complications, 560; diagnosis, 560; duration, termination, and prognosis, 561; treatment, 561.
  Pyelitis and Pyelonephritis, 562; history and bibliography, 562; etiology, 562; anatomical changes, 566; symptomatology, 570; diagnosis, 574; duration, termination, and prognosis, 575; treatment, 575.
  Nephrophthisis, Caseous Inflammation of the Kidneys, the Renal Pelves, and the Ureters, 577; introductory remarks, history, 577; bibliography, 578; etiology, 578; pathological anatomy, 579; symptomatology, 581; diagnosis, 584; duration, course, prognosis, 585; treatment, 586.
  Peri- and Paranephritis, 586; definition, history, and bibliography, 586; etiology, 587; pathological anatomy, 590; symptomatology, 593; complications and sequelæ, 597; diagnosis, 597; duration, termination, and prognosis, 600; treatment, 601.
Inflammatory affections, tendency to, in renal disease, 148.
Inflammatory processes in pyelitis, 572; in renal cirrhosis, 455; in etiology of fatty degeneration of kidneys, 603; extension of, in etiology of perinephritis, 588; of suppurative nephritis, 546.
Injections of warm solutions of salt into veins in ischæmia, 244.
Injuries in etiology of cancer of the kidney, 659; of movable kidney, 767; of perinephritis, 587; of suppurative nephritis, 545; of thrombosis of the renal vein, 782.
Inspection in general symptoms of renal disorders, 5.
Intermittent fever in etiology of amyloid degeneration of kidneys, 614.
Interstitial Inflammation or Induration of the Connective Tissue of the Kidneys. See Renal Cirrhosis.
Intestines, perforation into, in nephrolithiasis, 720; in perinephritis, 591; of renal abscesses, 558.
Iodine in nephrophthisis, 586; in vomiting of renal cirrhosis, 493.
Iron in amyloid degeneration of kidneys, 537, 633; in chronic parenchymatous nephritis, 407; in nephrophthisis, 586; in pyelitis, 577; in renal cirrhosis, 493.
Ischæmia of the Kidneys and its Results. The Choleraic Affections of the Kidneys, 222; etiology, 222; the morbid anatomy of the kidney in cholera, 226; pathology, 229; symptomatology, 233; diagnosis and prognosis, 242; treatment, 243.

Jaccoud, 171.
Jacksch, 573.
Jacobson, 130, 474.
Jaffé, 17.
Jaksch, 114.
Jaundice in cancer of kidney, 677.
Jerzykowsky, 660, 667, 675.
Jobert, 753.
Johnson, George, 177, 185, 193, 246, 258, 280, 324, 339, 456, 460, 463, 550, 615, 616, 618, 625.
Juergensen, 114.
Joints, affections of, in etiology of amyloid degeneration of kidneys, 612; changes in, in chronic parenchymatous nephritis, 348.

Kaltenbach, 574, 575, 593.
Kardell, 286, 289.
von Kaup, 114.
Kekulé, 497, 611.
Kelsch, 185, 187, 227, 229, 231, 233, 270, 325, 335, 349, 351, 353, 456.
Key, Axel, 83, 84, 86, 349.
Klebs, 44, 201, 271, 281, 344, 345, 346, 453, 519, 568, 569, 570, 622, 637, 638, 643, 662, 665.
Kletzinsky, 613.
Koenig, 546, 658, 743.
Koster, 642.
Koster, W., 778.
Krimes, 55.
Kuechenmeister, 743, 746, 747.
Kuehne, 119, 130, 157, 497, 611, 622.
Kupffer, 642, 762, 778.
Kussmaul, 123, 145, 214, 329, 614.

Laennec, 747.
von Langenbeck, B., 469, 778.
Lardaceous Kidney. See under Degenerative Processes of the Kidneys.

Lead, acetate of, in hæmaturia following cancer of kidney, 684; in pyelitis, 576.
Lead-poisoning in etiology of renal cirrhosis, 414.
Lebert, 582, 600, 659, 662, 665, 666, 678, 744.
Lecorché, 157, 183, 202, 248, 324, 352, 418, 419, 456, 504, 509, 511, 516, 537.
Lehmann, J. C., 39, 730.
Lennepveu, 558.
Leubuscher, 226, 228.
Leudet, 783.
Lever, 304.
Liebermeister, 136, 221, 415, 457.
Liebig, 26, 702.
Ligamenta rotunda, pain in, in suppurative nephritis, 553.
Lime, carbonate of, formations in nephrolithiasis, 697; oxalate of, formations in nephrolithiasis, 700.
Lindwurm, 244, 613.
Lipoma of Kidney. See under Tumors of the Kidneys.
Lipsky, 550.
Lithia, carbonate of, in nephrolithiasis, 734.
Little, 244.
Litzmann, 307, 310, 314, 321, 322.
Liver, changes in, in amyloid degeneration of kidneys, 552, 629; in cancer of kidney, 677; disease of, in etiology of suppurative nephritis, 546.
Lobb, 7[illegible]0.
Louget, 55.
Ludwig, 12, 375.
Luecke, 747.
Lundberg, 584.
Lungs, affections of, in amyloid degeneration of kidneys, 629; in cancer of kidney, 677; in chronic parenchymatous nephritis, 343, 348; in perinephritis, 597; in renal cirrhosis, 419, in etiology of passive hyperæmia of kidneys, 200.
Lymphangioma of Kidney. See under Tumors of the Kidneys.
Lymphatic tumors of Kidneys. See under Tumors of the Kidneys.

Maas, 657.
Magendie, 690, 730.
Magnesia, bicarbonate of, in nephrolithiasis, 735.
Malarial poisoning in etiology of chronic parenchymatous nephritis, 328.
Malgaigne, 739.
Malmsten, 170.
Manzolini, 660.
Marantic influences in etiology of thrombosis of the renal vein, 782.
Marcet, 687, 699, 703.
de Marchettis, Dominicus, 739.
Mascagni, 737.
Mayer, 270.
Mayer, Johann, 168.
Mayer, L., 249.
Measles in etiology of acute parenchymatous nephritis, 252.
Meckel, 176, 211, 497, 558, 580, 581, 611, 614, 616, 691, 700, 721, 777, 778.
Mental disturbances in uræmia, 110.
Meoli, 551.
Mercury in perinephritis, 601; in etiology of chronic parenchymatous nephritis, 329.
Meschede, 780.
Mesué, 763.
Meyer, Ludwig, 227, 228, 230, 233, 236, 237, 239.
Michaelis, 302.
Microscope, use of, in diagnosis of renal disease, 72.
Micturition, frequent, in renal cirrhosis, 418.
Middeldorpff, 647.
Milk cure in chronic parenchymatous nephritis, 406.
Mineral waters in nephrolithiasis, 734.
Modrzejewski, 611, 623.
Moeller, 764, 773.
Mohr, 583.
Monro, 769.
Moore, 675.
Morgagni, 705, 776.
Mosler, 13, 564, 583, 584.
Moxon, 638, 782.
Mucous membranes, affections of, in amyloid degeneration of kidneys, 522, 629; affections of, in etiology of amyloid degeneration of kidneys, 499.
Mueller, Joseph, 701, 702.
Mueller, Koloman, 255, 298.
Muenchmeyer, 780.
Muenzel, 619, 620, 621.
Mueller, Julius, 613.
Munck, Ph., 123, 621.
Munk, 618.
Murchison, 627, 725.
Muscles, fistulous tracts between, in perinephritis, 591; psoas, disease of, in etiology of suppurative nephritis, 546.
Musk in passive hyperæmia of kidneys due to heart disease, 217.
de Mussy, Gueneau, 774.
Myxoma of Kidney. See under Tumors of the Kidneys.

Nasse, 85.
Naunyn, 743, 747.
Necrosis of Kidney. See under Degenerative Processes of the Kidneys.
Neisser, 213.
Nephritis, Suppurative. See under Inflammations of the Kidney.
Nephrolithiasis. See under Foreign Bodies in the Kidney.
Nephrolithiasis in movable kidney, 771.

Nephrophthisis. See under Inflammations of the Kidney.

Nervous symptoms in amyloid degeneration of kidneys, 628; in pyelitis, 572.

Nessler, 130.

Neubauer, 12, 21, 30, 69.

Neuenahr, waters of, in nephrolithiasis, 734.

Neufoille, 777.

Neupauer, 692.

Niemeyer, 217, 406, 667, 669.

Nitre, in etiology of suppurative nephritis, 545.

Nothnagel, 82, 144, 192.

Number, Abnormal, of Kidneys. See under Anomalies in the Position.

OBERMÜLLER, 52.

Oedmansson, 83, 514.

Oertel, 84, 126, 250, 251, 253, 271.

Ogle, 556, 561.

Ogle, J. W., 719.

Ollivier, 413, 565, 781.

Operative measures in echinococci of the kidney, 756; in hydronephrosis, 657; in nephrolithiasis, 739; in perinephritis, 602.

Oppler, 116, 118.

Oppolzer, 764, 883.

Osborne, 111, 121, 149, 168.

Oxalate of lime formations in nephrolithiasis, 700.

Oxalic acid diathesis in etiology of nephrolithiasis, 690.

PAIN in symptomatology of renal disorders, 4; in acute hyperæmia of kidneys, 193; in acute parenchymatous nephritis, 273; in cancer of kidney, 670; in echinococci of the kidney, 749; in hemorrhagic infarction of kidneys, 606; in hydronephrosis, 653; in movable kidney, 769; in nephrolithiasis, 709; in nephrophthisis, 582; in perinephritis, 593; in pyelitis, 571; in renal cirrhosis, 420, 476; in suppurative nephritis, 552.

Palisade Worm. See under Strongylus Gigas, in Animal Parasites of the Kidneys.

Pallas, 742.

Palpation in general symptoms of renal disorders, 6.

Panum, 30, 323.

Paralysis in echinococci of the kidney, 751; in suppurative nephritis, 559.

Paranephritis. See under Inflammations of the Kidney.

Parasites of the Kidneys. See Animal Parasites of the Kidneys.

Parasites in etiology of perinephritis, 588; of pyelitis, 563.

Parenchymatous Nephritis, 244.

ACUTE PARENCHYMATOUS NEPHRITIS, 244; history, 244; etiology, 246; general summary of course and symptoms, 262; pathological anatomy, 265; analysis of symptoms, 273; prognosis, 288; diagnosis, 292; treatment, 295.

ACUTE PARENCHYMATOUS NEPHRITIS OF PREGNANCY, 304; etiology, 304; course, 311; pathological anatomy, 314; symptomatology, 316; prognosis, 322; diagnosis, 323; treatment, 323.

CHRONIC PARENCHYMATOUS NEPHRITIS, 324; etiology, 325; general course of the disease, 336; pathological anatomy, 343; symptomatology, 353; duration, 380; prognosis, 381; diagnosis, 393; treatment, 397.

Paulicki, 780.

Pendleton, 554.

Pentastomum Denticulatum of the Kidneys. See under Animal Parasites of the Kidneys.

Percussion in general symptoms of renal disorders, 8.

Pereverseff, 644.

Pericarditis in amyloid degeneration of kidneys, 629.

Perinephritis. See under Inflammations of the Kidney.

Peritonitis in amyloid degeneration of kidney, 514; 629; in movable kidney, 770; in nephrophthisis, 584; in perinephritis, 591; in renal abscess, 558.

Perl, Leopold, 465.

Perls, 118.

Petit, J. L., 600.

Petroff, 118.

Phlegmonous processes in etiology of fatty degeneration of kidneys, 603.

Phosphates, triple, formation of, in nephrolithiasis, 697.

Phosphatic diathesis in etiology of nephrolithiasis, 689.

Phosphorus poisoning in etiology of fatty degeneration of kidneys, 603.

Phthisis in etiology of amyloid degeneration of kidneys, 614; in nephrophthisis, 578.

Phymosis in etiology of pyelitis, 563.

Piorry, 728, 749.

Pirogoff, 593.

Pleurisy in amyloid degeneration of kidneys, 629; in perinephritis, 597.

Pneumonia in amyloid degeneration of kidneys, 629; in etiology of acute parenchymatous nephritis, 257.

Poland, 782.

Pollack, 783.

Ponfick, 252, 266, 271, 280, 283.

Portal, 558.

Portal, Antoine, 610.

Position, Abnormal, of the Kidney. See under Anomalies in the Position.

Potassium, borotartrate of, in amyloid degeneration of kidneys, 633; carbonate of, in nephrolithiasis, 724; iodide of, in amyloid degeneration of kidneys, 537; iodide of, in renal cirrhosis, 490; neutral salts of, in passive hyperæmia of kidneys due

to heart-disease, 218; nitrate of, in etiology of acute hyperæmia of kidneys, 192.
Pregnancy in etiology of acute parenchymatous nephritis, 304; of chronic parenchymatous nephritis, 326; of movable kidney, 767.
Pressure in etiology of thrombosis of the renal vein, 782.
Prognosis. See different diseases.
Prostate gland, enlargement of, in etiology of pyelitis, 564.
Prout, 36, 38, 40, 169, 710, 712.
Psoas muscle, affections of, in etiology of perinephritis, 597; of suppurative nephritis, 546.
Pus in albuminuria, 31; in ischæmia, 237; in renal cirrhosis, 418.
Pyæmic processes in etiology of fatty degeneration of kidneys, 603; of etiology of suppurative nephritis, 546.
Pyelitis. See under Inflammations of the Kidney.
Pyelitis in etiology of perinephritis, 588.
Pyelonephritis. See under Inflammations of the Kidney.

RAHN, 597, 599.
Ranke, Johannes, 730.
Rayer, P., 75, 97, 164, 167, 173, 185, 245, 258, 304, 310, 329, 414, 544, 546, 548, 554, 558, 562, 612, 636, 644, 658, 667, 695, 739, 763, 765, 766, 771.
Reaction, chemical, of urine, 23.
Récamier, 757.
von Recklinghausen, 606.
Rectum, perinephritis following operations upon, 589.
Rees, Owen, 112, 121, 170, 701, 712.
Rehder, J., 99, 102, 366.
Reinhardt, 174, 177, 182, 226, 228.
Relapsing fever in etiology of acute parenchymatous nephritis, 252.
Renal Abscess. See under Inflammations of the Kidney.
Renal Calculus. See under Foreign Bodies in the Kidney.
Renal Cirrhosis, 408; etiology, 409; progress of the disease, 415; pathological anatomy, 451; analysis of the symptoms, 462; *retinitis albuminurica*, 476; duration of genuine contracting atrophy of the kidneys, 480; prognosis, 482; diagnosis, 484; treatment, 488.
Renal Cysts and Cystic Degeneration of Kidney. See under Tumors of the Kidney.
Renal Gravel. See under Foreign Bodies in the Kidney.
Renal Vessels, Diseases of, 780; bibliography, 780.
DISEASES OF THE RENAL ARTERY, 780; pathology, 780.
DISEASES OF THE RENAL VEIN, 781; etiology, 782; general description, 783.
Respiratory organs, perforation into, of echinococci of the kidney, 751; in perinephritis, 591; in renal abscess, 558.
Rest in acute parenchymatous nephritis, 295; in chronic parenchymatous nephritis, 402; in hæmaturia following cancer of kidney, 684; in perinephritis, 601; in renal cirrhosis, 490; in suppurative nephritis, 561.
Retention of urine, in etiology of suppurative nephritis, 545.
Retinitis albuminurica in renal cirrhosis, 476.
Rheumatism, acute articular, in etiology of acute parenchymatous nephritis, 258.
Rickets, in etiology of amyloid degeneration of kidneys, 613.
Ricord, 536.
Rilliet, 692, 721.
Rindfleisch, 187, 202.
Riolan, Johannes, 763.
Roberts, 511, 512, 534, 578, 585, 657, 670, 671, 681, 704, 745, 755, 779.
Robin, Albert, 146, 157, 170, 665, 667.
Roetheln in etiology of acute parenchymatous nephritis, 252.
Rokitansky, 168, 176, 497, 610, 613, 614, 666.
Rollet, 673, 764, 766, 771.
Rommelaere, W., 124, 126.
Rose, Cooper, 650.
Rosenstein, 7, 9, 21, 56, 111, 120, 130. 181, 203, 305, 309, 318, 320, 322, 408, 414, 514, 565, 573, 589, 680.
Rovida, 83, 84, 90, 380.
Rudneff, 497, 611, 622.
Ruehle, 622, 623.
Ruysch, 644.

SALGADO, 711.
Salicylic acid in pyelitis, 576.
Salzbrunn, waters of, in nephrolithiasis, 734.
Sandwith, H., 776.
Sarcoma of Kidney. See under Tumors of the Kidneys.
Scanzoni, 313.
Scarlatina in etiology of acute parenchymatous nephritis, 247; of chronic parenchymatous nephritis, 326.
Scheele, 687, 699.
Scherer, 211, 655, 690.
Schiff, 55.
Schlossberger, 695.
Schmalfuss, 754.
Schmidt, C., 106, 611.
Schneider, 760.
Schorer, 329.
Schottin, 115, 116, 341.

Schroeder, Karl, 668, 669.
Schroff, 247.
Schueppel, 666, 683.
Schultzen, 690.
Schuster, 554.
Sclerosis, Renal. See Renal Cirrhosis.
Scrofula in nephrolithiasis, 721; in etiology of amyloid degeneration of kidneys, 499, 612.
Scurvy in etiology of pyelitis, 563.
Seegen, 735.
Seeligsohn, 700.
Ségalas, 690, 731.
Senator, 39, 40, 43, 510, 534, 631.
Senna in acute parenchymatous nephritis, 300.
Septic processes in etiology of fatty degeneration of kidneys, 603.
Serous cavities, affections of, in chronic parenchymatous nephritis, 348.
Sex in etiology of amyloid degeneration of kidneys, 616; of cancer of the kidney, 662; of chronic parenchymatous nephritis, 336; of hydronephrosis, 645; of movable kidney, 765; of nephrolithiasis, 692; of nephrophthisis, 578; of perinephritis, 589; of renal cirrhosis, 411; of renal cysts, 640.
Sexual organs, affections of, in the female, in etiology of pyelitis and pyelonephritis, 564.
Sexual power, loss of, in renal cirrhosis, 422.
Siebert, 547, 559.
von Siebold, 743.
Siemon, 21.
Simon, 16, 104, 454, 469, 654, 657, 711, 727, 739, 740, 757, 763.
Simpson, 737.
Sinapisms in etiology of acute hyperæmia of kidneys, 191.
Skin, affections of, in amyloid degeneration of kidney, 534; in chronic parenchymatous nephritis, 348; in perinephritis, 595; in renal cirrhosis, 422, 426; in etiology of amyloid degeneration of kidneys, 499.
Small-pox in etiology of acute parenchymatous nephritis, 252; of fatty degeneration of kidneys, 603; of perinephritis, 589.
Soda, phosphate of, in nephrolithiasis, 733; sulphate of, in nephrolithiasis, 734.
Solon, Martin, 304.
Somnolence in uræmia, 110.
Sowloff, N., 71.
Spectroscope in diagnosis of hæmaturia, 70.
Spiegelberg, 119, 753, 754.
Spiroptera Hominis. See under Animal Parasites of the Kidneys.
Spleen, changes in, in amyloid degeneration of kidneys, 522, 629; chronic parenchymatous nephritis, 348.
Stanley, 559.
Steiner, 647, 661, 765.
Stern, 762.
Stewart, Grainger, 105, 177, 182, 183, 414, 453, 457, 460, 461, 482, 511, 512, 517, 518, 519, 534, 611, 625, 631, 633.
Stimulants in amyloid degeneration of kidneys, 537; in ischæmia, 244; in nephrophthisis, 586; in passive hyperæmia of kidneys due to heart-disease, 217; in renal cirrhosis, 493.
Stoehr, August, 496.
Stokvis, 17, 36, 37, 38, 41, 54, 55.
Stomach, diseases of, in nephrolithiasis, 722.
Strauch, 119, 130, 157.
Strength, loss of, in renal cirrhosis, 422.
Stromeyer, Louis, 244.
Strongylus Gigas of the Kidneys. See under Animal Parasites of the Kidneys.
Sugar in urine in ischæmia, 239.
Sulphur in nephrolithiasis, 732.
Sulphuric acid in pyelitis, 576; poisoning by, in etiology of fatty degeneration of kidneys, 603.
Suppuration in amyloid degeneration of kidney, 629; in etiology of acute parenchymatous nephritis, 253; of amyloid degeneration of kidneys, 498, 612; of chronic parenchymatous nephritis, 326.
Suppurative nephritis. See under Inflammations of the Kidney.
Sutton, Henry, G., 184, 409, 410, 457, 459, 460, 461, 463.
van Swieten, 720.
Sydenham, 720, 731, 733.
Symptomatology. See different diseases.
Symptoms, general, of renal disorders. See General Symptoms of Renal Disorders, 3.
Syphilis in etiology of amyloid degeneration of kidneys, 499, 612.

Taester, 625.
Tannin in hæmaturia following cancer of kidney, 684; in pyelitis, 576.
Tarasp, waters of, in nephrolithiasis, 735.
Tar water in nephrolithiasis, 732.
Tanchou, 660.
Taylor, 557, 627.
Tegard, 38.
Temperature of body in acute parenchymatous nephritis, 273; in amyloid degeneration of kidneys, 627; in cancer of kidney, 677; in nephrolithiasis, 710; in nephrophthisis, 584; in perinephritis, 594; in pyelitis, 571; in suppurative nephritis, 552.
Tenner, 123, 145, 214.
Testicles, pain in, in nephrolithiasis, 710; in suppurative nephritis, 553; affections of, in etiology of perinephritis, 588.
Thênard, 737.
Thirst in renal cirrhosis, 422.

Thomas, 82, 237.

Thompson, 735.

Thrombosis of renal veins following amyloid degeneration of kidneys, 523.

Todd, 325, 556, 611, 627, 669, 726.

Toël, 702.

Transfusion of blood in renal cirrhosis, 496.

Traube, 121, 124, 132, 135, 137, 149, 157, 175, 180, 181, 186, 202, 207, 214, 246, 320, 343, 367, 368, 409, 418, 456, 463, 464, 465, 467, 569, 576, 606, 607, 611, 625, 626, 630, 712.

Travers, 711.

Treatment. See different diseases.

Treitz, 114, 573.

Treyden, 553.

Triple phosphate formations in nephrolithiasis, 697

Troja, 553, 559, 666, 680, 698, 711, 739, 766.

Trousseau, 587, 590, 596, 599, 772.

Tubercles of the Kidneys. See under Tumors of the Kidneys.

Tuberculosis in etiology of amyloid degeneration of kidneys, 499, 612; of nephrolithiasis, 721; in pyelitis, 563.

Tuengel, 435, 646.

Tumor, presence of, in cancer of kidney, 671; in nephrolithiasis, 716; in nephrophthisis, 583; in perinephritis, 594; in suppurative nephritis, 555.

Tumors of the Kidney, of the Pelvis of the Kidney, and of the Perinephritic Tissue, 635; bibliography, 635; introductory remarks, 635.

  RENAL CYSTS AND CYSTIC DEGENERATION OF THE KIDNEYS, 638; bibliography, 638; *cyst-formations in extra-uterine life*, 639; *congenital cysts*, 641.

  HYDRONEPHROSIS, 643; bibliography, 643; history, 644; etiology, 644; pathological anatomy, 648; symptomatology, 651; complications and diagnosis, 654; duration, termination, and prognosis, 656; treatment, 656.

  CANCER OF THE KIDNEY, 658; bibliography, 658; history, 658; etiology, 658; pathological anatomy, 662; symptomatology, 670; complications, 677; diagnosis, 678; duration, course, prognosis, 683; treatment, 684.

  LYMPHATIC NEOPLASMS, 685.

  TUBERCLES OF THE KIDNEYS, 685; bibliography, 685; pathology, 686.

Turpentine in nephrolithiasis, 732; in etiology of acute hyperæmia of kidneys, 192; of pyelitis, 563.

Typhoid fever in etiology of acute parenchymatous nephritis, 253, 257; in etiology of perinephritis, 589; of pyelitis, 563.

Typhus fever in etiology of perinephritis, 589; of pyelitis, 563.

ULCERS of soft parts in etiology of amyloid degeneration of kidneys, 613.

Unruh, Carl Oscar, 293.

Uræmia accompanying renal diseases, 109; in acute parenchymatous nephritis, 284; in acute parenchymatous nephritis of pregnancy, 313; in amyloid degeneration of kidneys, 518; in cancer of kidney, 675; in chronic parenchymatous nephritis, 369; in hydronephrosis, 651; in renal cirrhosis, 425, 473.

Urea, examination for, 26.

Ureters, changes in, in etiology of hydronephrosis, 647.

Urethra, stricture of, in etiology of pyelitis, 561.

Uric acid diathesis in etiology of nephrolithiasis, 639, 694.

Urine, changes in, in acute parenchymatous nephritis, 274; in acute parenchymatous nephritis of pregnancy, 316; in amyloid degeneration of kidneys, 505, 624; in cancer of kidney, 674; in chronic parenchymatous nephritis, 353; in echinococci of kidney, 751; in fatty degeneration of kidneys, 604; in ischæmia, 234; in movable kidney, 771; in necrosis of kidneys, 608; in nephrolithiasis, 705, 711; in nephrophthisis, 581; in perinephritis, 596; in pyelitis, 570; in renal cirrhosis, 431, 462; in suppurative nephritis, 553; ammoniacal decomposition of, in etiology of pyelitis and pyelonephritis, 563; retention of, in etiology of suppurative nephritis, 545; examination of, in general symptoms of renal disorders, 10; suppression of, in acute hyperæmia of kidneys, 193.

Urinous smell in renal cirrhosis, 429.

Ustimowitsch, 14.

Uterus, changes in, in etiology of hydronephrosis, 645; perinephritis following operations upon, 588.

VALENTIN, 170, 174.

Varandaeus, Joh., 732.

Vauquelin, 687.

Venereal excesses in etiology of suppurative nephritis, 547.

Venous stasis in etiology of fatty degeneration of kidneys, 603.

Vertebral column, gravitation of pus along the, in perinephritis, 591.

Vertigo in renal cirrhosis, 418.

Vessels, renal, diseases of. See under Renal Vessels, Diseases of.

Vichy, waters of, in nephrolithiasis, 734.

Vierordt, 17.

Vigla, 75.

Virchow, 64, 176, 178, 182, 186, 187, 226, 315, 352, 373, 497, 517, 519, 544, 611, 612, 615, 619, 627, 637, 642, 646, 659, 660, 685, 695, 696, 697.

Voelckers, 421, 426, 437, 455.
Voelckers on Retinitis Albuminurica in Renal Cirrhosis, 276.
Vogel, 12, 14, 15, 28, 367, 555, 582.
Voigtel, 548.
Voit, 126, 127, 132, 147, 150.
Vomiting in acute parenchymatous nephritis, 286; in amyloid degeneration of kidneys, 513; in cancer of kidney, 676; in movable kidney, 769; in nephrolithiasis, 710; in perinephritis, 594; in pyelitis, 572; in renal cirrhosis, 422, 425; in renal diseases, 152; in uræmia, 111.

Wagner, E., 589, 614, 666, 685, 697, 758.
Waldeyer, 549, 614, 664, 638, 696, 698, 747, 773.
Walshe, 658, 665.
Walter, Friedrich August, 644, 698.
Walther, 764.
Warmth in perinephritis, 602.
Wasting of muscles and fat in renal diseases, 151.
Waters, mineral, in nephrolithiasis, 734; plain, in nephrolithiasis, 731.
Waxy Kidney. See under Degenerative Processes of the Kidneys.
Webb, Allan, 692, 613.
Weber, O., 146.
Weigert, C., 551.
Weisbach, 762.
Wells, 34, 35, 245, 334.
Wells, Spencer, 650, 653, 655, 663, 680.
Werlhof, 72.
Wertheim, 603.
Whyttel, 743, 746.
Wiederhold, 557.
Wieger, 320.
Wildungen, waters of, in nephrolithiasis, 735.
Wilks, Samuel, 324, 392, 615.
Willigk, 661.
Wilson, 658.
von Wittich, 36, 55.
Woehler, 702, 734.
Wolcott, 684.
Wolff, 744.
Wollaston, 687, 701.
Wollner, Gustav, 256.
Wood, 581.
Wundt, 37.
Wyss, 235, 236, 237, 238, 239, 243, 747.

Xanthine formations in nephrolithiasis, 703.

Yellow fever in etiology of acute parenchymatous nephritis, 253.

Zalesky, 119.
Zenker, 668.

297274

MPr
Z

Author Ziemssen, Hugo Wilhelm von (ed.)

Title Cyclopaedia of the practice of medicine. Vol.15.

University of Toronto
Library

DO NOT
REMOVE
THE
CARD
FROM
THIS
POCKET

Acme Library Card Pocket
Under Pat. "Ref. Index File"
Made by LIBRARY BUREAU